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SYMPOSIUM ON OTOLARYNGOLOGICAL PROBLEMS IN SEPSIS.

I.—PARAPHARYNGEAL INFECTIONS AND INTERNAL JUGULAR VEIN THROMBOSIS: DIAGNOSIS AND TREATMENT.*

DR. AUGUST L. BECK, New Rochelle, N. Y.

In writing this paper considerable repetition of what was written in a previous paper[1] has been unavoidable. The exact diagnosis of deep neck infections necessitates a consideration of the site of initial infection or portal of entrance, a thorough understanding of the applied anatomy and a familiarity with the regional manifestations which can be more or less grouped anatomically.

The most common cause or starting point of neck infections is inflammation in or about the tonsils. This includes the pharynx. Dental infection, particularly in the mandible, is fairly common. Nasal sinusitis is a frequent forerunner of neck infection. Other less common causes are operations on the tonsils or adenoids, trauma to the pharynx, hypopharynx or esophagus, infection in the thyroid gland or its fascia, infection from the middle ear by way of

*Read as part of a Symposium before New York Academy of Medicine, Section on Otolaryngology, Oct. 18, 1933.

the auditory tube, infection from the mastoid called Bezold's abscess, infection from the petrous temporal bone, infection at the base of tongue and floor of mouth, caries of the cervical

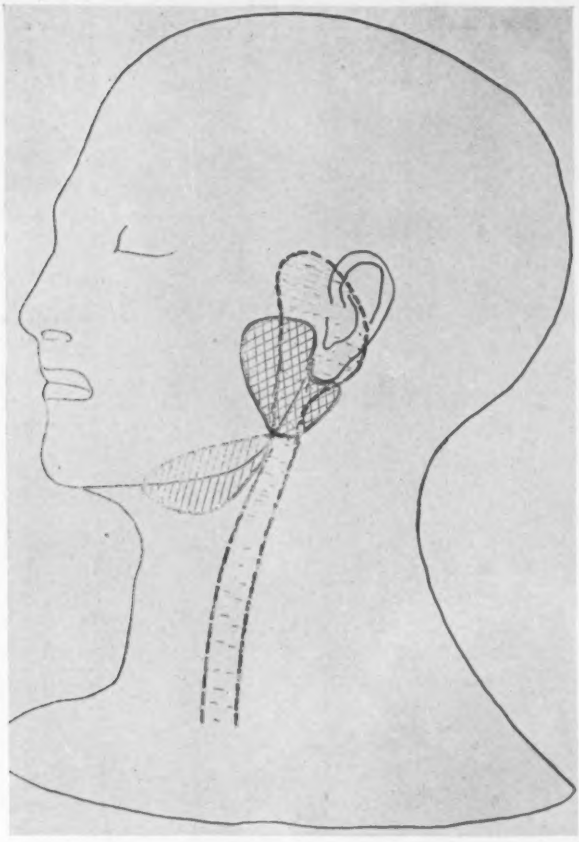

Fig. 1. Fascial compartments in the upper part of the neck.

vertebrae, thrombosis of the pterygoid plexus of veins, external neck injuries, fractures of the larynx and trachea, perforation of the esophagus, suppurating cysts, and fractures and injuries of the jaw. Any inflammation that can throw infec-

tion into a cervical lymph node may thereby be the cause of a neck infection.

APPLIED ANATOMY.

In considering the applied anatomy of the cervical fascia, all its related structures must be included, as the clinical

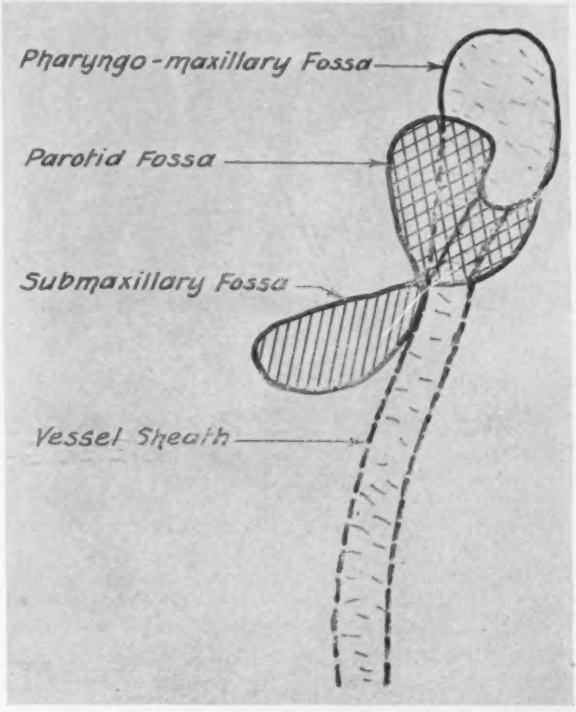

Fig. 2. Relationship of these compartments to each other.

picture, local and general, when inflammation is present, is determined and colored by involvement of these structures.

The fascia exists to bind the structures of the neck together, holding them in their allotted places, and separating them

from one another. Because of the significance of symptoms and signs resulting from involvement of these structures when inflamed, I consider mention of them important. They are the lymphatic vessels and nodes, the blood vessels, the nerves from which symptoms are not so rare, the cervical viscera and muscles which form the framework for the fascia, and which give enveloping attachment to its layers.

Clinically the commonest symptoms arise from the lymphatics and veins. General sepsis is the prompt result of severe involvement of either or both. High continuous temperature can be accounted for by lymphatic involvement, with or with-

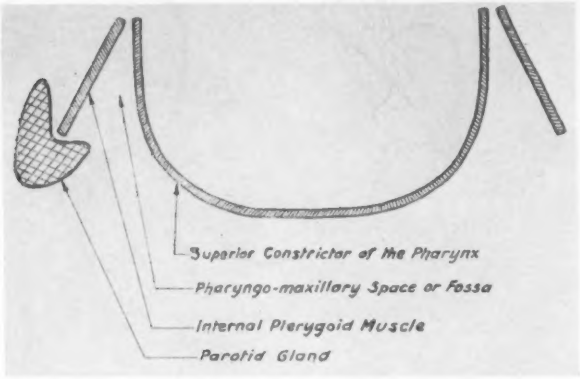

Fig. 3. Diagrammatic cross-section drawing of the pharyngomaxillary fossa.

out visible adenopathy. Abrupt drops and elevations of temperature with chills are evidences of venous or blood stream involvement. Trismus and torticollis are evidences of muscle splinting. Paralyses affecting the pupils, pharynx, palate, tongue, shoulders and larynx are evidences of nerve involvement. Hoarseness, dyspnea and dysphagia are evidences of visceral involvement.

Mosher's[2] work on the applied anatomy of the cervical fascia is the foundation of all that is here set down. His conception that all the layers of the deep cervical fascia may be regarded as offshoots from the carotid sheath is superbly simple. In his inimitable way he calls the sheath the "Lincoln highway

of the neck," and states that it is the highway of travel for pus and for the search of pus. There are three large compartments in the upper part of the neck which communicate more or less directly with the sheath of the great vessels.

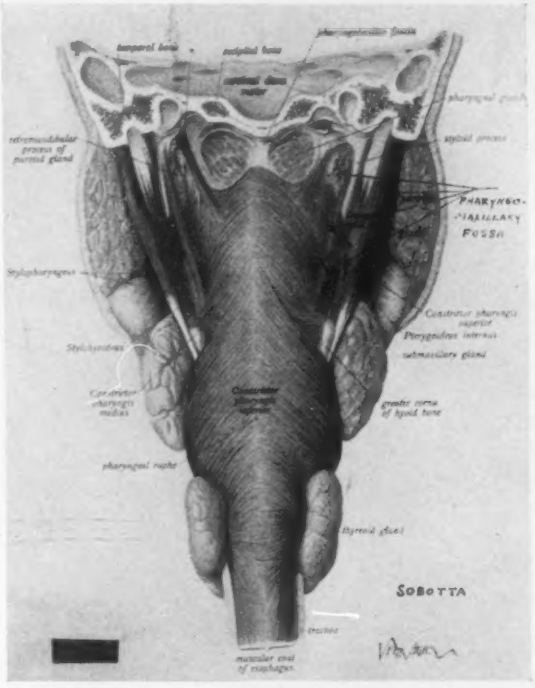

Fig. 4. The pharyngomaxillary fossa as seen from behind.

These are the parotid, the submaxillary and the pharyngomaxillary. I shall demonstrate these on lantern slides.

REGIONAL MANIFESTATIONS.

Grouping of neck infections according to their particular regional manifestations is an attempt at classification, anatomical and clinical. In an analysis of cases from which I derived my experience, they were found to fall definitely into the following groups:

1. Pharyngomaxillary space infection.
2. Carotid sheath infection.
3. Submaxillary space infection.
4. Prevertebral fascia infection.
5. Pretracheal, buccopharyngeal or visceral fascia infection.
6. Cervical gland infection.

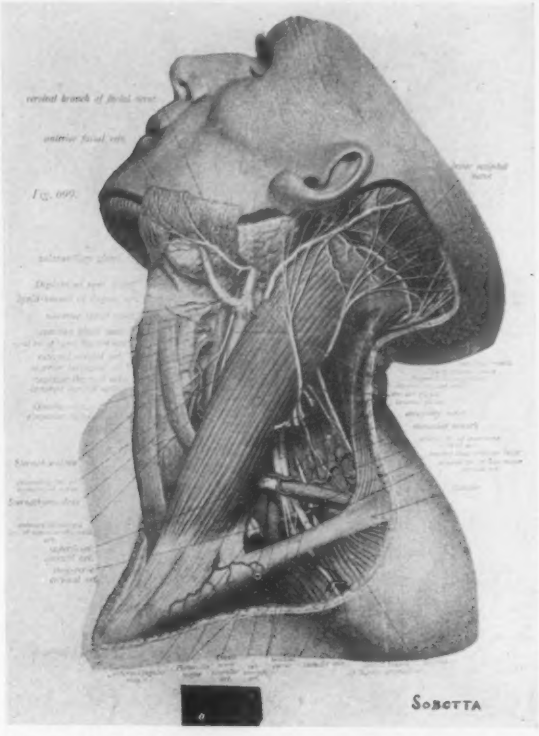

Fig. 5. Close relationship of the parotid and submaxillary glands to each other and to the carotid sheath.

All except the last of these groups conform to a definite anatomical grouping. A separate grouping of cervical adenitis is considered desirable and necessary because any cervical gland infection may be the forerunner of a deep fascia infection, and should be closely watched lest a severe sepsis sud-

denly occur. I have not yet observed an infection which has remained strictly confined to the posterior cervical triangle. When it has been so limited it was classified under cervical gland infection because it was strictly an adenopathy. Extension immediately puts it into one of the other groups.

I have been asked how it is determined into which group a case should be placed. If seen early, the portal of entrance

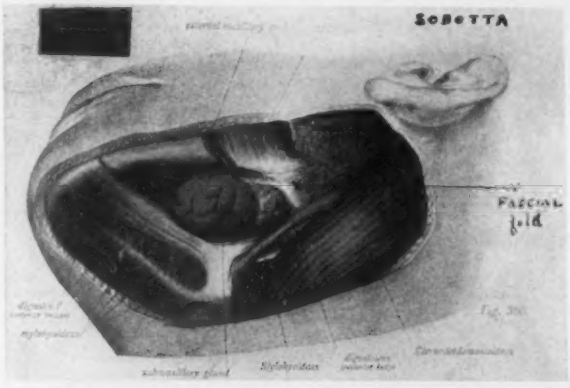

Fig. 6. Fold of fascia between the two salivary glands where the superficial and deep layers unite at the jaw angle.

and etiology are of considerable help. With this knowledge, one may anticipate which compartment is likely to be invaded. The actual involvement of a particular compartment is demonstrated by certain signs and symptoms. Extension from the compartment first invaded to one or more other compartments can then be followed. Close observation of the behavior of the lymph nodes will give valuable clues. The site of initial involvement of the neck with recognition of extensions will point where drainage is needed. A careful inspection should be made at least once or more times daily; also a daily blood count and frequent blood culture examinations.

PHARYNGOMAXILLARY FOSSA INFECTION.

This is the compartment most frequently involved from infections high up, *e.g.*, in the tonsils, nose, sinuses,

pharynx and auditory apparatus. The patients frequently come referred with a diagnosis of quinsy sore throat. Involvement should be considered present if there is trismus and swelling of the lateral pharyngeal wall with or without change of color. There may be prolapse or displacement of the tonsil without any inflammation of the tonsil itself. Swelling over the region of the parotid gland may be considered additional evidence as it indicates secondary invasion of the parotid compartment. Infections below the level of the angle of the jaw do not give these signs. The trismus is caused by splinting of the internal pterygoid muscle which forms part of the outer wall of the space. Chills not only indicate immediate

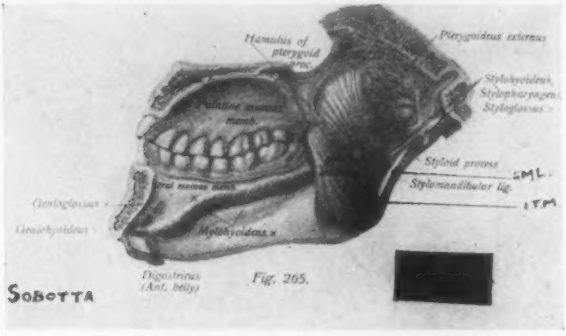

Fig. 7. Stylomandibular ligament and internal pterygoid muscle.

drainage of the pharyngomaxillary compartment, but if they continue, also ligation or resection of the internal jugular vein. Where the infection has been caused by a deeply located peritonsillar abscess tonsillectomy may bring about a prompt cure. If after tonsillectomy there is a bacteremia, indicated by chills and a positive blood culture, external drainage of the pharyngomaxillary space, and ligation or resection of the internal jugular vein should be done.

CAROTID SHEATH INFECTION.

This, as can be explained by the applied anatomy, is secondary to infection of any or all of the other compartments and their contained lymph nodes. A particularly noteworthy cause

is infection, with or without suppuration, of the lymph nodes located on the vessel sheath itself. Evidence that the sheath and its contents are infected is chiefly general in character, and may be expressed in one word, *viz.*, sepsis. The picture is much the same as it is where the sigmoid sinus is infected during the course of a mastoid inflammation. If the blood cultures are positive, all doubt is eliminated; if negative, and

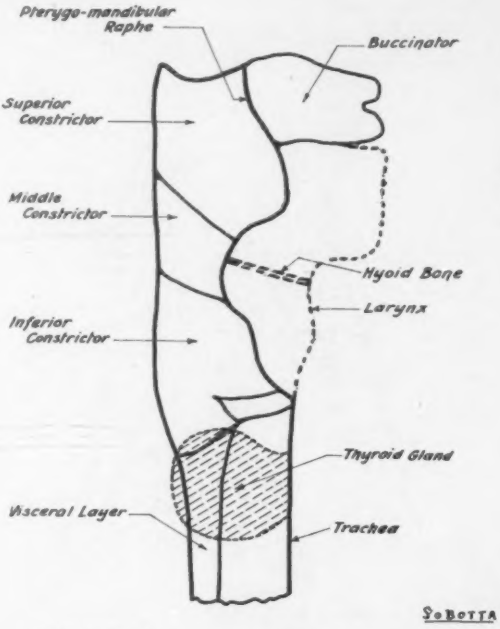

Fig. 8. Skeleton outline of the visceral fascia.

sepsis continues, drainage is indicated. *The degree of sepsis is the one great guide.* One should not wait for positive blood culture, as the culture may be negative even when the blood is withdrawn from the internal jugular vein itself. The sudden appearance of painful inflammatory swelling in the calf of the leg, in the foot or pain in a joint is strong evidence of blood stream infection, and calls for immediate drainage of the neck and ligation of the vein. If this is done promptly,

the secondary lesions may recover without surgical intervention.

Local evidence of infection of the sheath is often lacking. Tenderness on palpation over the sheath and deep to the sternomastoid muscle is an unreliable sign of vein infection; so

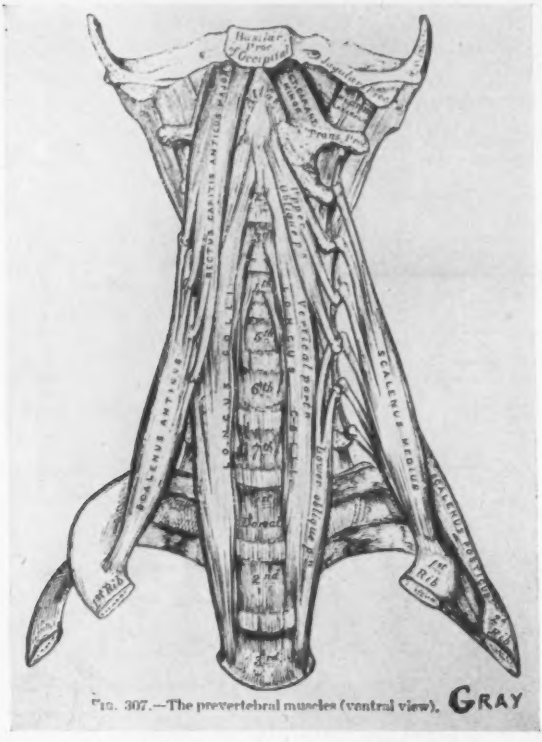

Fig. 307.—The prevertebral muscles (ventral view).

Fig. 9. Bony and muscular framework upon which the prevertebral fascia is reflected.

also is swelling, for both may be caused by inflamed lymph nodes, and not necessarily by inflammation of the vein wall. Diminishing swelling of the neck over and about the sternomastoid muscle should not be construed as contraindicating surgical drainage if the sepsis continues. My most danger-

ous cases were of this type. Torticollis toward the opposite side is a most positive sign of inflammation under the sternomastoid muscle.

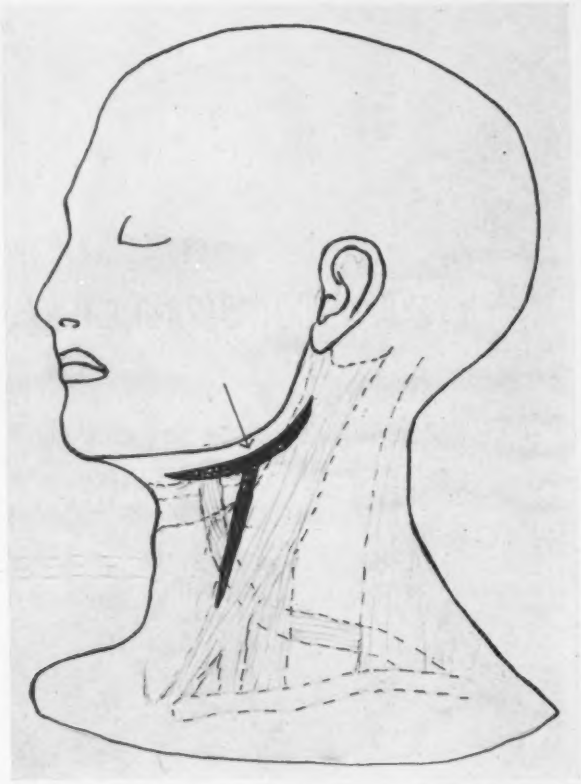

Fig. 10. Mosher's incision for exposing the submaxillary gland and draining the pharyngomaxillary fossa

SUBMAXILLARY FASCIA INFECTION.

This group is rather common, and is frequently designated Ludwig's Angina. It may be divided into two groups, one of which is caused by dental infection, and the other not.

Cases of Dental Origin: In this group there are two very pronounced signs, *viz.*, swelling of the submaxillary and sub-

mental regions and extreme trismus. There is usually swelling and induration of the floor of the mouth, gums and tongue, and a foul odor. These cases usually give a history of trouble in the teeth at the onset, and frequently date from the time

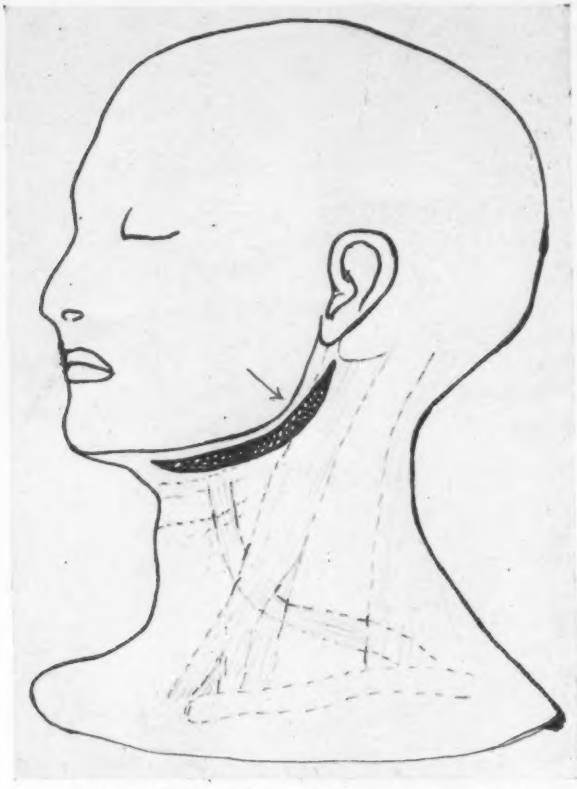

Fig. 11. Incision through which the stylomandibular ligament may be reached beneath the angle of the jaw.

teeth were extracted. In this connection it is possible that the injection of the local anesthetic may be the direct provocative in some cases. Inflamed gums should not be injected. They yield promptly to submaxillary drainage, and can usually be given a good prognosis. Severe dental pathology may be

taken care of at the time that the neck is drained. Osteomyelitis of the mandible sometimes occurs, but external drainage of the neck protects the cervical fascia, and prevents blood stream infection.

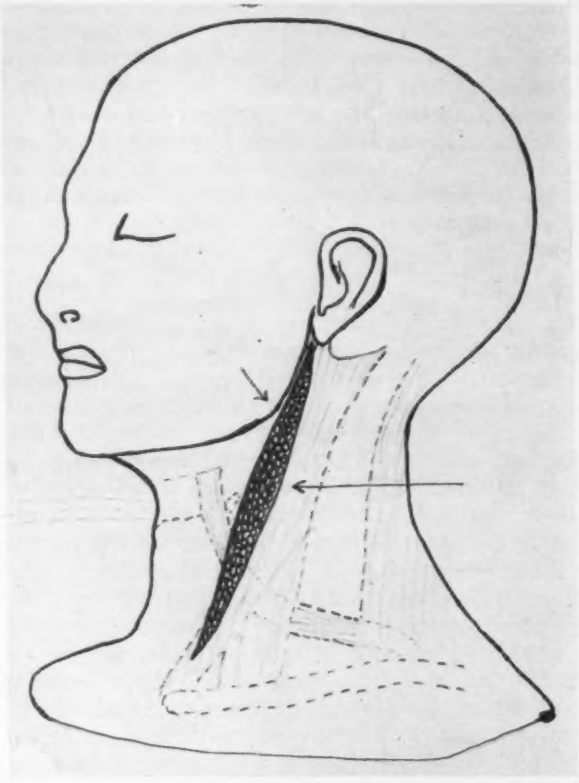

Fig. 12. Incision through which the stylomandibular ligament may be palpated and the internal jugular vein resected; also for Dean's approach to a retropharyngeal abscess.

Cases of Nondental Origin: Those not of dental etiology are dangerous and insidious. Infections in the uppermost part of the respiratory tract, pharynx, tonsils, and base of the tongue, are the usual starting points, and the lymphatics

are regarded as the connecting link. Frequently the first swelling indicating a neck infection occurs in the submaxillary triangle. The inflammation rarely remains confined to the submaxillary space, but extends into the parotid space or into the pharyngomaxillary space or both, and downward below these into the lower part of the neck along the vessel sheath. The applied anatomy of the submaxillary and submental triangle regions suggests that infection would find a barrier to direct downward extension in the midregion of the neck at the hyoid bone where the fascia gets a firm attachment. Therefore, deflection laterally to the vessel sheath is to be expected if extension is not checked by drainage. I have several times observed cases to go on to jugular vein thrombosis, in which the submaxillary swelling had almost completely subsided.

PREVERTEBRAL FASCIA INFECTION.

This is commonly termed retropharyngeal abscess. It most frequently occurs in infants and young children, and the symptoms and signs are too well known for detailed enumeration. Infection in and about an adenoid, suppuration of the uppermost postpharyngeal lymph nodes, and caries of the cervical vertebrae are the most common causes. A true midline retropharyngeal abscess tends to extend downward along the fascia of the longus colli muscles so that when it reaches the hypopharynx, the swelling protruding forward, caps the larynx and seriously embarrasses respiration. When a patient with a typical retropharyngeal abscess is seen before respiratory embarrassment has occurred, peroral incision is usually promptly effective. If not, Dean's[2] external route for drainage is an excellent and easy method of approach. Where there is already severe dyspnea from respiratory obstruction, it may be necessary to first relieve this by tracheotomy, particularly if there is considerable exhaustion. Radiographs can be of invaluable diagnostic assistance.

PRETRACHEAL, BUCCOPHARYNGEAL OR VISCERAL FASCIA INFECTION.

This layer is of importance because of its contained viscera. Infections which penetrate from the hypopharynx and esophagus involve this layer; also those infections in and about

the larynx, trachea and thyroid gland. Infection following trauma from bronchoscopy or esophagoscopy is an example. When the infection is high up, lateral to the naso and oropharynx, it becomes a pharyngomaxillary space infection. Symptoms and signs of involvement will vary with the location of the infection, which ranges from the middle constrictor of the pharynx and hyoid bone down to the chest. Moderate diffuse swelling of the side or midline of the neck anywhere from the level of the hyoid bone to the clavicle may be present. Infection penetrating from one side of the hypopharynx causes swelling lateral to the thyrohyoid and laryngeal regions. A suppurating thyroglossal cyst is midline in location. Infection of the thyroid gland fascia sometimes strikingly resembles a goitre. Infections resulting from esophageal trauma are the most dangerous and profound of all neck infections, and chest involvement usually occurs before there is any local evidence in the neck. Hoarseness, aphonia, dyspnea, odynphagia and dysphagia are symptoms indicating visceral involvement. Hoarseness in one case may be due to inflammatory swelling in and about the larynx, and in another, to paresis of a recurrent laryngeal nerve.

Infection in and about the hypopharynx may sometimes be drained by internal incision, working through the laryngeal or esophageal speculum. Surprisingly large collections of pus may thus be evacuated. If drainage is good, and the patient not septic nor dyspneic, external drainage may be avoided; but if the infection persists, or the patient becomes septic or dyspneic, external drainage should be done without delay. This type of case should be watched for downward extension, for involvement of the vessel sheath, and for laryngeal and tracheal obstruction.

CERVICAL GLAND INFECTION.

There is a sharp distinction between inflammation of the superficial and inflammation of the deep glands.

Inflammation of the Superficial Glands: The superficial glands lie between the superficial fascia and the superficial layer of the deep fascia, and drain into the glands of the deep fascia. When inflamed, suppuration with abscess formation is a common result, and in such instances there is no demonstrable involvement of the fascial planes other than that which

is present at the place where the glands are suppurating. It is then strictly localized and in fact the suppuration brings about localization. In such cases, incision should be delayed until frank pus may be evacuated; that is, until fluctuation or inflammation of the skin is present, provided that there is no sepsis and no evidence of deep involvement. Suppuration of the superficial glands is the only type of neck infection where delay until fluctuation is present is a safe procedure. Sometimes there is involvement of the deep glands and fascia after the superficial gland abscess has been incised and drained.

Inflammation of the Deep Glands: Often in these cases the individual glands cannot be felt, and there is no localized suppuration. There may be only a slight fullness along the sternomastoid muscle, making that side of the neck diffusely prominent. There is well marked tenderness on pressure or manipulation of the muscle due to inflammation of the nodes beneath it which lie on the carotid sheath. In these, suppurative disintegration of the glands takes place late or not at all, and sepsis and blood culture examinations become the important indicators. Spontaneous recovery is possible and common if until then there has been no sepsis. Once a definite sepsis has given evidence of itself, surgical drainage is indicated. A mild non-alarming sepsis may simmer along for several weeks when suddenly there is an explosion ushered in by a chill or by a distant metastasis. Constant watching is necessary. Thrombosis of the internal jugular vein because of failure to recognize this dangerous condition is the cause of many a fatality.

Inflammatory swelling of the deep glands is very commonly seen in two localities, namely—the region centering at the bifurcation of the common carotid artery, and in the submaxillary triangle. Other localities are—upper part of the occipital triangle, the uppermost postpharyngeal glands, the lymph nodes in the parotid and submaxillary compartments, and nodes in the thyroid gland fascia.

TREATMENT.

Remarks on treatment will be limited to a description of surgical approaches for drainage which will be demonstrated on the lantern slides.

CONCLUSIONS.

A discussion of complications has been omitted. The occurrence of a distant complicating infection may be regarded as a positive indication for immediate surgical drainage of the neck, and in my experience also resection of the internal jugular vein. If this is done promptly there often occurs spontaneous subsidence of such a secondary manifestation. Painful, tense, inflamed swelling of the calf of the leg, dorsum of the foot, or joint pain, usually occurring suddenly, are examples of such complications. In conclusion, I wish to call attention to the fact that the absence of free, visible pus in a compartment at the time of operation is very common, and does not justify the conclusion that infection is not present. I have seen a macerated, necrosed vein with completely obstructing thrombus with no visible pus. A permanent remission of temperature and cessation of sepsis follow adequate drainage. In cervical fascia infections drainage placed in advance of the involvement is better than drainage which only trails the involvement. The late Dr. John Mackenty fully realized this in the postoperative care of laryngectomy cases. Finally, thrombosis of the internal jugular vein is the cause of the sepsis which terminates the life of these patients when drainage is delayed, and is often unrecognized.

BIBLIOGRAPHY.

1. Beck, August L.: A Study of Twenty-four Cases of Neck Infection. *Trans. Amer. Acad. Ophth. and Otolaryngol.*, pp. 342-381, 1932.

2. Mosher, Harris P.: The Submaxillary Fossa Approach to Deep Pus in the Neck. *Trans. Amer. Acad. Ophth. and Otolaryngol.*, pp. 19-36, 1929.

3. Dean, L. W.: The Proper Procedure for External Drainage of Retropharyngeal Abscess Secondary to Caries of the Vertebrae. *The Jour. of Laryngol., Rhinol. and Otolaryngol.*, Sept., 1918.

421 Huguenot Street.

SYMPOSIUM ON OTOLARYNGOLOGICAL PROBLEMS IN SEPSIS.

II.—NEWER CONCEPTIONS IN THE MANAGEMENT OF SEPTIC SINUS THROMBOSIS.*

DR. O. JASON DIXON, Kansas City, Mo.

After hearing all these very interesting discussions that so frequently mentioned the ligation of the internal jugular vein, I am very much in the same position as the minister about whom the late Calvin Coolidge told Mrs. Coolidge when she didn't accompany him to church one day. You probably have all heard the story. Mrs. Coolidge asked him what the minister talked about. He said, "Sin." She said, "What did he have to say about it?" Coolidge said, "He said he was against it."

Well, I am against this ligation for thrombosis of the sigmoid sinus, and it is better to say thrombophlebitis than thrombosis. To get back to the beginning of this treatment, I find that it started with the first recognition of the lesion, and the same operation with the same technique has been carried on ever since. Zaufal along in 1880 was the first man to discover a thrombus; also, if we will recall from our medical history, those were the days when they were operating and taking off arms and legs for infections. I believe this procedure is a carry-over of the false hope that we may be able to head off an infection by tying the vein ahead of it. I am very glad not to hear tonight anything said about embolism because I am firmly convinced that it does not exist except in our imagination. That left the door open for controversy, whether to ligate the vein before the sigmoid sinus or after.

I am not going into a discussion of the presentation of clinical cases. I do not know anything more misleading or less conclusive than to try to prove that more cases, or this

*Read as part of a Symposium before New York Academy of Medicine, Section on Otolaryngology, Oct. 18, 1933.

one case recovered because I tied the internal jugular vein, or that one did not recover because I did not tie the vein. I was formerly sold on the proposition and I have tied a great number of veins. I would try for a while to tie one and leave another untied on patients coming in at the same time. I was convinced that ligation had nothing whatever to do with the recovery. I admit it is not a serious operation. It does not add a great hazard to the operative risk and is easily done, but it is not based on sound pathological principles. In the first place, nature was apparently aware of the fact that she would get into trouble with this sigmoid sinus because she produced ideal conditions for blocking off this vein. The slowing and intermittent interruption of the blood stream and eddying of the blood lend to the production of a thrombus. All one has to do is to uncover the sigmoid sinus at operation, and allow the patient to come out from a deep anesthesia and take a deep inspiration. If the vessel is not thrombosed you can see it collapse. In other words, the blood is being sucked out faster than it can fill. It is the only place in the body where a large venous channel comes almost directly into contact with the outside. In other words, I like to interpret a mastoiditis as a compound fracture. Infection travels from the Eustachian tube to the middle ear and invades the mastoid cells. It is then directly in apposition with this large venous channel. Through this travels an enormous amount of blood from the brain with every ideal arrangement for the production of a thrombus.

Why did nature want to produce a thrombus? Because thrombosis is one of the best protective mechanisms we have, protection from invasion of the blood stream and protection against the loss of this vital fluid. It is a wonderful phenomenon that this blood can change from a liquid to a solid state. Frequently that is all that is necessary. However, unfortunately, this stagnant thrombus, this clot, within the vessel becomes a foreign body when it becomes a thrombus. It is no longer body tissue and it is therefore more susceptible to infection. This thrombus I take it, is more like a detour. These blood platelets, these red blood cells and white blood cells and this fibrin sacrifice themselves to this lesion.

The phlebitis that occurs in mastoiditis I believe is almost always due to direct invasion from infection of the overlying

bone. I routinely expose the sigmoid sinus because I so frequently find it involved and because I feel that it does no harm. This particular area of the sinus is furthest from the dura. Rarely do we get into any trouble in exposing the sigmoid sinus and allowing pus to run over. We so often find necrosis and liquifaction of the inner table of the bone without any apparent symptoms that we are led to believe it is a common occurrence, and phlebitis is the first protective mechanism that takes place. A thrombus is formed and it may remain a mural thrombus. If the bone is removed early enough that is frequently all that happens. The thrombus may extend up or down, but once that blood stream is completely occluded the formation of the thrombus then is always retrograde.

It is very interesting to look back over the old literature. I ran across an article by the veterinarian Williams, an Englishman, who found thrombosis in horses was always retrograde. I found the same thing in cattle. On lowering their heads they were unable to drink or eat with the head down. All these things he described. He talked it over with the pathologist Spence in London. Spence showed him the same thing in his specimens. It is only natural for it to form that way because this thrombus is like a dam in a stream, the debris is always on the upper side. The thrombus is retrograde. Now we cannot tie above. Neither can we tie the superior or inferior petrosal veins. It is a large task and entails a great deal of unnecessary risk to dissect out the jugular bulb, to leave part of the infected vein and try to ligate above the torcular end. In other words, we lock the back door and leave the front door open. The real way to prove this is to follow these patients to necropsy, but our observations as to what happens to the patients who recover is purely speculative. We should live long enough to follow them to necropsy and see what happens, as Dr. Mackenzie did with his heart cases.

I decided it was necessary for me to do something for these patients and I started to work experimentally on the dog. The dog is a wonderful experimental animal. It more closely simulates the human than any other animal. I started a series of experiments three years ago, looking primarily for a substitute for the gauze packing in the thrombus. In using the gauze pack we do not know when to pull it out. Frequently

it is too early and we pull the thrombus out with the gauze, and in repacking, the patient had one chill and I had the other one. Then I left it in too long and the patient had a chill. I thought this was due to an infection from the packing. That is not good surgery, to put in a packing through an infected wound. I tried all kinds of things and finally hit on the idea that I could use the patient's own tissue. All that is necessary is to lengthen the primary mastoid incision and follow it down over the sternocleidomastoid muscle. Leaving it attached, section a piece of muscle the length you desire. Open the sigmoid sinus and remove the thrombus if you desire, and lay this muscle over it. It seals it off. It not only acts as a fine dressing, but it also brings in a fresh additional blood supply and also a fresh lymphatic supply, which I think is important, to the infected area. It quickly produces thrombosis. Dr. Mills, of the University of Cincinnati, has been doing some very wonderful work on thrombosis and he finds that muscle is very rich in fibrinogen. We do not thoroughly understand all we say about blood clotting. We know we have thrombin and prothrombin, etc. I do not want to get into that tonight. Muscle does stimulate thrombosis and a thrombus is formed, in addition to controlling the bleeding and bringing in a fresh blood supply. Another thing is that the infected and damaged vein can be left alone. The ideal situation is to remove the infected thrombus without going through the vein, but we must go through the vein of course to get the thrombus. What do we get in return in repacking after removal of the infected thrombus? We get a new thrombus which we trust will not become infected. There must be a thrombus there whenever a vessel is cut. It is part of the normal healing. But if we leave an infected foreign body such as gauze there, the thrombus which is formed, as I said, is nonviable tissue. It becomes infected and we simply repeat the process.

We find also in the dog that when I put this muscle on the external jugular vein (the external jugular is the larger vein in the dog), or if I put it in the vein or bring the ends together in the vein or run a loop through, always using viable muscle, the vein always recanalizes and returns to its normal size as a blood carrier. I have had no human patients come to necropsy so that I cannot say what happens there. I will show you some X-rays on these dogs later. I have a

method of diagnosing a thrombus or an obstruction in the vein, using an opaque media. I was led into that field because I did not want to sacrifice the dogs or the specimens. I did not want to repeat all that hard work. Every time I took out a specimen, that stopped that experiment and I had to start anew. I admit that I have not proven to you men who believe in ligation that it is wrong to ligate, that it is not good, that it is not founded on sound physiological or pathological reasoning, but I hope to when I finish and show these lantern slides on the dog. The dog tells his own story. It shows the recanalization and the direction of the thrombus which will help clear my point.

You may wonder why I did not use the sigmoid sinus in the dog. The dog has a small brain compared to the human, and his blood supply goes principally to the muscles of mastication and the salivary glands. So I cannot simulate that picture because it is not anatomically possible. However, prior to doing this operation I did take dogs and experiment by ligating the internal jugular and external jugular veins and the carotid arteries to get the normal physiological changes. I have also been able to produce thrombosis and phlebitis, but not with the bacteria that are so destructive to humans. It was possible only with the organism peculiar to the dog, the organism of distemper. I do not know what that organism is. If I did I would be lecturing before the Veterinary's Society. Someone told me that if I discovered the organism of distemper it would be more profitable than if I found a cure for cancer. I have been side-tracked into belly surgery, spleen surgery, aneurysms and all kinds of wild things in this work, but one must always try to keep from going down side alleys. It all has some relation to this problem.

We must always bear in mind that infections do not travel by one route. Infections of the upper lip may reach the sinus by the ophthalmic vein, but by no means does that infection stay on the "Lincoln Highway" exclusively. In the dog I can by the X-rays show these little fine venules, these subcutaneous veins that develop in the dog as a means of compensating for the ligation of the internal and external jugular veins. So we are dealing with things that are frequently beyond our control, and I believe we are going to get further with these infections of the head and neck by standing by

and not getting into too big a hurry to tie something or try to head off something. I know the pressure is great. It comes from the relatives and family. They want something done, not just transfusions and forced fluids. By the way, I do not know of any better treatment for septic sinus thrombosis than blood transfusions and forced fluids. There isn't much pain in this condition. The digestive tract is all right, the kidneys are all right. I do not enjoy these suppurative head infections and I am very happy to see a suppuration in the soft tissues. That patient is vaccinating himself. We should not be too afraid of pus or too much in a hurry to drain it. It is dead tissue and a fine method of vaccination. My experience is that these patients, if it does not strike the viscera, the lungs, spleen, heart or kidneys, and stays peripheral, practically always recover. The unfortunate thing about ligation again is that those things have already happened before you ligate the vein. It is really too late. I still insist it is too late unless we do it as a routine procedure in all our ear infections.

1103 Grand Avenue.

SYMPOSIUM ON OTOLARYNGOLOGICAL PROBLEMS IN SEPSIS.

III.—DISCUSSION OF THE BASIS FOR THE SELECTION OF THE TYPE OF PROCEDURE IN SINUS THROMBOSIS.*†

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In the recent literature on otitic sinus thrombosis, the old controversy between the school favoring jugular ligation and that advocating jugular resection has been replaced by a new thought emanating from a third and new school which favors the deletion of all surgery to the internal jugular vein in the management of otitic sepsis. From time to time reports of individual cases have appeared in which an otitic sepsis with a demonstrable thrombus recovered as a result of thrombectomy alone. Recently, however, more extensive data has appeared, based upon observations of cases in series, which has given weight to the claims of those who would discard all surgery to the internal jugular vein in cases of otitic sepsis.

Among the more important contributions on this subject are those of Portmann,[11] Krepuska,[1] Hirsch,[2] Mygind,[3] Heine,[4] Botey,[5] Körner,[6] Gruenberg,[7] Halle, Jansen,[8] Knutson[9] and most recently Dixon.[10] The conclusions reached by these men can be classed into three groups: Those favoring ligation in all cases, those reserving the procedure for selected instances, its use being determined by the clinical picture and blood studies, and those who claim that the internal jugular vein should never be operated upon in any case of septic sinus thrombosis.

In the discussion of the surgical therapy of any condition. one must be guided by basic anatomy, basic pathology and clinical experience. In the matter of septic sinus thrombosis, all otologists are unanimous in advocating the removal of the

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thrombus from the lateral sinus. It is only in the utilization of jugular ligation that opinions differ. In order to arrive at a logical conclusion anent jugular ligation and resection it is therefore necessary to study certain fundamental facts.

Anatomy: While the jugular vein is the direct continuation of the lateral sinus, it must be borne in mind that other venous channels also communicate with the lateral sinus and so afford other routes for the spread of infection. These consist of the condyloid veins, the inferior and superior petrosal sinuses, the marginal sinus and the mastoid emissary vein. Those who would discard ligation in sinus thrombosis advance, as one of their arguments, the fact that ligation of the internal jugular vein in no way influences the spread of the infection into the general circulation because in itself it does not remove the possibility of a spread through the other channels. On the other hand, those who favor jugular ligation advance an equally potent point in that they maintain that, by ligation, the main channel for spread is thus shut off. Both these contentions are justified, but from the anatomical standpoint alone a decision cannot be reached. For, given a case wherein there is present within the sigmoid sinus alone a mural thrombus or thrombus undergoing disintegration at its distal extremity, and wherein the tributaries to the bulb are patent, it is certain that infected material will follow the natural path of the blood and be carried into the internal jugular vein. In this instance, then, a thrombectomy alone will not suffice to clear the sepsis because the blood from the inferior petrosal will wash over the infected wall of the bulb and continue the supply of infection to the general circulation. On the other hand, where any of the tributaries are also the seat of an infected thrombus, the ligation of the internal jugular vein will not influence the course of the sepsis.

Pathology: The essential point to remember in dealing with otitic sinus thrombosis is that the cause of the sepsis is not the thrombus, but the thrombophlebitis which is invariably present. An inflammatory lesion is present in the vessel wall which, in its fully developed stage, involves all the layers. Within this wall bacteria are present. This phlebitis, in the majority of cases, precedes the formation of a thrombus. The thrombus is a manifestation of the body's attempt to defend itself against the invasion of the bacteria into the general

circulation. Consequently, in the type of sinus thrombosis following the coalescent and the chronic mastoidal suppurations, the thrombus becomes infected secondarily. This infection occurs at its center while its extremities remain, for a time, sterile. Where the thrombus within the sinus is the result of an extension of a thrombus from a smaller vessel into it, such as occurs in late cases of hemorrhagic mastoiditis, a phlebitis of the inner layer of the wall alone is present, and the thrombus is more apt to be a mural clot infected throughout. In either event, it must be borne in mind that the phlebitis is as much responsible for the sepsis as the thrombus is. In the early stages of development it is the phlebitis alone that causes the clinical picture of sepsis and may even be responsible for a positive blood culture through the ingress of bacteria through the vasa vasorum. Only later, when the thrombus within the vessel begins to disintegrate, or wherein an infected mural clot is present which is washed by the blood stream, does the thrombus enter into the picture as a cause for the continuance of the sepsis. Whether the growth of the thrombus is retrograde, that is toward the torcula, or whether it grows in the direction of the blood stream, matters little as far as the etiology of the general sepsis is concerned, as long as the ends are sterile and no disintegration has occurred. A thrombus, presenting a sterile extremity to the remaining open circulation, cannot infect the general blood stream until it disintegrates and permits the infected material or the bacteria themselves to gain access to the circulation. Several times, in the writer's own experience, the removal of just such a type of thrombus, namely, an obliterating clot showing organization at both ends and where, after removal, bleeding was obtained from the superior petrosal, the torcular end of the sinus and from the bulb, the sepsis continued unabated until a wide exposure of the infected wall of the sinus showed the phlebitis to extend into the bulb. Only after thoroughly curetting the infected bulb and ligating the internal jugular was the sepsis controlled.

Consequently, to discard jugular ligation or resection because it does not influence the retrograde growth of a thrombus is not logical. If one is careful in the management of the proximal end of the exposed sinus to remove every vestige of infected wall in addition to obtaining free bleeding from that end before plugging off, that end of the sinus will give no

further trouble. On the other hand, to remove the septic thrombus *in toto* and to permit an infected wall to be present and untouched within the bulb and in addition fail to expose the internal jugular to determine whether or not this structure shows a phlebitis is, in the writer's opinion, poor surgical judgment. For, since the lateral sinus and the internal jugular vein are in reality one continuous channel, it is impossible to determine accurately, without actual inspection, whether or not a severe bulbar phlebitis or even a thrombosis has not extended to and involved the jugular vein.

Clinical Data: When one compares the results of the various surgeons who have reported the efficacy of the treatment of sinus thrombosis with and without jugular ligation, it is seen that in different hands different results are obtained. The percentage of cures with ligation varies from 0 to 85 per cent, whereas without ligation from 29 to 100 per cent (Botey, who reports four cases). Braun[12] states "no definite conclusions can be drawn from these statistics. They are of no value because no attention is paid to the different malignancy in different cases and to indications for ligation in various cases." It is an axiom of medicine that each case is an entity in itself though it may be part of a general group of cases. In the writer's experience, both on his own cases and those of his fellow staff members, it was found that no general rule could be formulated for the surgical management of all cases of sinus thrombosis. In certain cases a simple incision of the sinus wall and removal of the thrombus sufficed for a cure of the sepsis. In others, a thrombectomy coupled with a Voss exposure of the bulb and a thorough curettage of the bulb resulted in recovery. In two instances a wide dissection of the horizontal portion of the lateral sinus was needed in order to excise a badly diseased sinus wall; this procedure was performed some time after the removal of a small obliterating thrombus at the knee and the establishment of free bleeding from both ends failed to cure the sepsis. In each case, after ten days, no clot had formed at the upper end and the subsequent exposure of the horizontal portion of the lateral sinus revealed an extensive phlebitis of the outer wall, which was not looked for at the original operation and which accounted for the continuation of the sepsis. This secondary operation caused a prompt cessation of the bacteremia and a cure of the patient. The internal jugular vein was not operated upon

in any of these instances cited because it was felt, from the findings at operation, that this structure was not involved.

On the other hand, in very many instances, the pathology at operation, namely, a diseased bulb, caused us to expose the internal jugular vein and either ligate or resect it, depending upon whether or not a phlebitis of the vein was present.

From the anatomic and pathologic facts and from his clinical experience the writer has come to the conclusion that no general rule can be formulated for the surgical management of all cases of sinus thrombosis. Unlike Dixon, who states: "I will never ligate another vein," the writer feels that each case must be judged for itself and the surgical therapy varied as the indications for its use are presented.

Indications for the Type of Surgery to be Employed in Sinus Thrombosis. Preventive: In the development of an otitic sepsis due to a septic thrombus in the sinus which follows in the course of a coalescent mastoiditis, the intravenous clot is always preceded by a phlebitis of the outer sinus wall. This phlebitis, in its earliest stage, involves only the outer layer of the vein wall and will subside once the pathology within the mastoid process has been removed and the diseased sinus plate removed. Where, however, the inflammation of the vein wall is extensive, and the outer wall very much thickened, the foregoing procedure does not always suffice to cause a cessation of the activity of the bacterial infection in the sinus wall. Too often, the simple mastoidectomy and the removal of diseased sinus plate covering an extensive phlebitis is followed, after an interval of time, by the appearance of an otitic sepsis. During the time elapsing between the operation and the onset of the sepsis, the phlebitis has extended to involve the inner layer of the sinus, with a resultant protective thrombus which soon undergoes disintegration. It is the writer's contention that, when an extensive phlebitis is found at the time of the mastoid operation, an excision of the entire area of the sinus wall which is diseased should be done then and there. The phlebitis of the vein wall is a pathological condition, which, if permitted to remain, may lead to further involvement of the sinus with the subsequent development of a bacteremia. No one would think of leaving diseased cells and granulation tissue undisturbed within the mastoid cavity proper; no operator would fail to remove a necrotic tegmen or sinus plate,

because, to leave these is to provide a nidus for further spread toward the endocranium. Yet, many surgeons permit and even advise leaving a badly diseased sinus wall untouched simply because no clinical signs of sepsis have as yet appeared. The writer[13] feels that it is far more logical to consider the phlebitis where it is severe, as pathology which should be removed, rather than to permit it to remain and be a potential source for the development of a sinus thrombosis with its attendant general sepsis. The excision of the diseased wall and the blocking off the sinus has, in the writer's experience, obviated many cases of otitic sepsis which otherwise might have developed, for no such complication has occurred in any of his cases since he adopted this procedure. Since one cannot definitely state which diseased sinus wall will eventually cause a sepsis to appear and which will not, it is far better to remove the widespread phlebitis in every case and so assure no further extension.

Septic Sinus Thrombosis with Obliterating Clot: 1. There are many cases of a thrombus within the sinus which are accidentally discovered during the course of a mastoid operation. These cases present no signs of sepsis. At operation a perisinal abscess is found covering a very much thickened and white sinus wall. Palpation of the wall elicits the transmitted pulsation of the intracranial vessels. On incision into the sinus, an organized, almost completely white thrombus is found or one undergoing organization. Such a finding indicates a thrombophlebitis which is in the process of healing or has already healed itself. The white sinus wall which presents no granulations on it is evidence that repair of the inflammatory lesion in the wall proper has been undertaken by connective tissue proliferation. The organization of the thrombus is also evidence of repair. Consequently, such a lesion affords no threat of a general sepsis and the management of the lesion can be twofold. The sinus can be left entirely alone and the clot undisturbed or through a simple incision of the outer sinus wall the thrombus can be removed with a forceps or suction and free bleeding established from both ends. The incision in the sinus wall can then be plugged. Surgery to the internal jugular need not be done in these cases.

2. Where, at operation, the sinus wall is covered with granulations, and where after removal of the granulations the

transmitted pulsations of the brain vessels are felt, the procedure to be followed depends upon several factors.

a. If no clinical or bacteriological evidences of sepsis are present the sinus wall should be incised before inserting any plugs. The insertion of plugs in any case where an obliterating clot is present distorts the findings within the sinus and makes it impossible to determine, when the clot is examined, whether or not its extremities are undergoing disintegration. The incision of the sinus wall where an obliterating clot is present will not be followed by bleeding and consequently plugging of the sinus is not needed for the prevention of bleeding. A sinus, after the granulations have been removed, will not pulsate unless an organized clot is present within it (Valenti,[14] Portmann[11]).

After incision of the sinus wall and the exposure of the clot, the vein wall should be inspected to determine the extent of the phlebitis and so much of the outer wall excised as is found involved. This should be done before the clot is disturbed. If the diseased wall is found to extend above the knee the bone should be removed and excision of the wall continued up to the normal wall. Leaving a portion of the diseased wall in contact with the blood stream after a plug has been inserted will not only result in failure of this end to clot and become obliterated, but will bring an infected area of sinus wall in contact with the blood stream and so may afford a factor in the production of a sepsis.

If the phlebitis is found to extend into the bulb, a simple exposure of the posterior portion of the bulb by the Voss technique will permit curetting this out after removal of the thrombus. The thrombus should next be removed with a forceps or suction, and the bleeding ends of the opened sinus blocked. No surgery to the jugular is here required.

b. If clinical evidences of sepsis are presented in the temperature curve, but wherein no chills or chilly sensations have occurred and where the blood culture is sterile and no marked secondary anemia is present, the same procedure should be followed out as outlined in *2a.* If after the excision of the diseased sinus wall, and the removal of the thrombus, free bleeding is obtained from both the bulbar and torcular ends

of the opened sinus, the ends should be plugged and no surgery to the jugular performed.

If, on the other hand, the removal of the thrombus cannot be accomplished *in toto* either from the distal or proximal ends, further measures are required. A suction tip, preferably a semi-soft rubber tube, should be inserted into the torcular end and an attempt made to remove the remainder of the clot in this manner until free bleeding occurs. If this is not successful, further removal of bone and exposure of the sinus, even to the torcula is indicated. If the superior petrosal also appears to be thrombosed the bone in the sinodural angle should be removed and the petrosal incised and the clot sucked out until bleeding occurs.

If no bleeding occurs from the bulbar end of the sinus and suction and curettage of the bulb after its exposure does not yield free bleeding, then, despite the negative blood culture and the absence of a bacteremia, the internal jugular vein should be exposed and inspected. If the phlebitis has extended to this vessel or if it is the seat of a thrombus, a wide resection of the diseased portion of the jugular is indicated. If the vessel is found normal in appearance it should be ligated. Whether the vein is here simply ligated or cut between two ligatures matters little as long as the ligation is made above the facial vein. This procedure guarantees one thing, namely that no infection will find its way into the general circulation through the main channel. That it does not in any way tend to favor a spread of the thrombus into the inferior petrosal has been demonstrated by Haymann and Torok. Where such a spread has already taken place into the inferior petrosal, further surgical procedures are at the present not available for attacking this structure.

c. Where the clinical evidences of sepsis are present, together with rigors, a positive blood culture and a rapidly increasing secondary anemia, there, in the writer's opinion, the safest procedure is a thorough surgical attack of the sinus, and bulb if necessary, and a ligation or a resection if indicated, of the internal jugular vein. During the past seven years each case of sinus thrombosis at the Beth Israel Hospital which came under this category was treated differently. In some instances only the sinus surgery was performed at first. In others, the internal jugular was exposed at the same

time as the sinus was opened. In only one case, already reported by Kopetzky,[17] did a patient having a positive blood culture recover without jugular surgery. In this instance the culture yielded only three colonies of streptococcus hemolyticus to the entire plate. All of our other cases in which an attempt was made to do without jugular surgery in the presence of a more marked bacteremia continued to show a stationary or increasing bacteremia and sepsis, and jugular ligation had to be resorted to before abatement of the sepsis was obtained. In three cases with positive blood culture operated upon during the past year, the internal jugular, when exposed at the time of the original operation on the mastoid and sinus, was found to be markedly thickened and discolored and the resected portion of the vein showed, on microscopic examination, an extensive phlebitis. Had these cases been subjected to a thrombectomy and excision of the sinus wall alone, the process in the jugular would have been overlooked and the cause of the sepsis not entirely removed.

From the experience at our hospital, therefore, the writer feels that where a demonstrable bacteremia is present, the internal jugular vein should be exposed in every case. The fact that some cases may recover without resort to jugular surgery does not outweigh the importance of removing all pathology which is responsible for the continuance of a sepsis, and to hope that the jugular is normal when a simple procedure is at hand to readily determine its condition, is not in accord with the best principles of therapy.

d. Where an obliterating thrombus has undergone disintegration to the extent that fluid pus is present within the sinus, or wherein the outer sinus wall has been entirely necrosed so that its visceral layer is open to the mastoid cavity, the procedure to be selected depends upon the clinical picture and blood studies.

Where no sepsis is present and the blood culture is negative, it can be safely assumed that nature has successfully walled off the suppurative process and limited it to the sinus. Here simple drainage of the sinus is all that is needed. It is unwise here to attempt to establish free bleeding and then plug off, for a natural blockage of the blood stream is already present.

On the other hand, where there is fever, regardless of its degree or of its character, and despite a negative blood cul-

ture, it is safer to ligate the jugular and establish free bleeding if, after three days, the simple drainage of the sinus has not resulted in a dropping of the temperature. In such a case we must reason that the continued fever is the result of a continuing inflammatory process, and since a portion of the thrombus has already disintegrated into fluid pus it is probable that the rest of the clot is also breaking down.

Primary Bulb Thrombosis: Here, the appearance of the sinus may afford no clue. It is the clinical picture coupled with the finding of a normal mastoid process and a positive blood culture upon which the indications for surgery are founded. Here, after exposure of the sigmoid portion of the sinus, the writer has extended his procedure by exposing the posterior aspect of the bulb after the Voss technique. The sinus is then plugged off proximally before opening into it. The distal end is not plugged. It is preferable in these cases to insert the upper plug below the entrance of the mastoid emissary vein so as to shut off the bleeding from this vessel. An incision is then made in the lower end of the sinus and extended into the bulb. If the clot is an obliterating one, no bleeding occurs until its removal and this end is then plugged off. If, as frequently happens, the clot is a mural one, curettage of the bulb must be rapidly performed in spite of the free bleeding. The excess blood is rapidly removed by mass suction and a thorough curettage can be accomplished in a few seconds. The internal jugular vein should next be exposed and either ligated or resected if it is also diseased.

Septic Sinus Thrombosis with a Mural Thrombus: This type of thrombus is found in the earliest stages of invasion of the sinus by extension from a coalescent or chronic mastoidal disease and in the thrombosis resulting from an extension of a thrombus from a small tributary into the lumen of the sinus proper, such as occurs in the late cases of thrombotic or hemorrhagic mastoiditis. The latter type of case is very frequent during epidemics of Spanish influenza. Here the same principles outlined for the management of the case with an obstructing thrombus should hold. Where a demonstrable bacteremia is present the sinus should be obliterated and the jugular exposed and treated. Where no bacteremia or metastasis are present, obliteration of the sinus and removal of the clot should be first performed and jugular surgery may

be reserved pending the influence of the first procedure on the clinical course of the case.

And finally, with our present knowledge concerning the management of sinus thrombosis and with excellent results being reported by various men using different measures, some always operating on the jugular, some never operating on it and others taking a course between these two, one cannot consent to discard surgery to the jugular entirely until more proof is brought forth and an exact analysis of each case is presented by the men advocating thrombectomy alone.

BIBLIOGRAPHY.

1. Krepuska, G.: Ueber otogene Pyämie mit besonderer Berücksichtigung der Frage der Jugularisunterbindung. *Ztschr. f. Laryngol., Rhinol.*, Vol. 17, p. 176, 1928.

2. Hirsch, M.: Zur Frage der Jugularisunterbindung bei Sinusphlebitis. *Ztschr. f. Laryngol., Rhinol.*, Vol. 17, p. 110, 1928.

3. Mygind, S. H.: Treatment of Otogenic Sepsis and Sinus Thrombosis. *Acta Otolaryngologica*, Vol. 16, p. 474, 1931.

4. Heine, B.: Operationen am Ohr, etc. S. Karger, Berlin, 1913.

5. Botey, R.: Faut-il ou ne faut-il pas lier la jugulaire dans la phlébite du sinus lateral? *Trans. 8th Internat. Otol. Congress*, p. 137, 1909.

6. Korner, O.: Die Otitischen Erkrankungen des Hirns, etc, 1925.

7. Gruenberg, G.: Zur Frage ueber Bedeutung der Unterbindung der Vena jugularis interna bei otogenen Pyemien. *Monatsschr. f. Ohrenheilk.*, Vol. 59, p. 289, 1925.

8. Jansen, A.: Ueber Hirsinusthrombose nach Mittelohreiterungen. *Arch. f. Ohrenheilk.*, Vol. 35, p. 55, 1893.

9. Knutson, E.: Treatment of Otogenic Thrombophlebitis. *Acta Otolaryngologica*, Vol. 6, p. 359, 1924.

10. Dixon, O. J.: Nonligation of the Internal Jugular Vein in the Treatment of Sigmoid Sinus Thrombophlebitis. *Trans. 38th Ann. Meeting Amer. Laryngol., Rhinol. and Otolog. Soc.*, p. 142, 1932.

11. Portmann, G.: Considérations sur le traitement de phlébites et thrombophlébites sinusojugulaires. *Rev. de Laryng.*, Vol. 52, p. 523, 1931.

12. Braun, A.: Sinus Thrombophlebitis. P. B. Hoeber, New York, 1928.

13. Almour, R.: Prevention and Minor Symptoms of Sinus Thrombosis. *Arch. Otolaryngol.*, Vol. 6, p. 373, 1927.

14. Valenti, S.: Valore clinico della pulsazione del seno transverso. *Boll. d. mal. d. orecchio, d. gola del. naso.*, Vol. 48, p. 129, 1930.

15. Beck, O.: Histo-pathologische Veränderungen am Sinus Sigmoideus des Menschen Hervorgerufen durch Kompression. *Acta Otolaryngolojica*, Vol. 12, p. 228, 1928.

16. Kopetzky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis. *Arch. Otolaryng.*, Vol. 10, p. 302, 1929.

17. Kopetzky, S. J.: Studies in Otitic Sepsis. *Ann. Otol., Rhinol. and Laryngol.*, Vol. 37, p. 329, 1928.

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SYMPOSIUM ON OTOLARYNGOLOGICAL PROBLEMS IN SEPSIS.

IV.—SUMMATION OF TREATMENT OF SEPSIS FROM THE MEDICAL STANDPOINT.*

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Unfortunately, we medical men have no specific treatment for general infections, whether due to the ear or whether the primary infection is elsewhere. The treatment lies mainly in the domain of adequate surgery. I am also glad that I am a medical man, so that I can pass over the controversy as to whether or not the jugular vein should or should not be tied. Dr. Dixon's work is certainly suggestive and he is contributing a great deal to our knowledge of thrombosis and the possibility of regeneration of the blood vessels.

It has been my good fortune to have been intensely interested for a long period of time in so-called otitic sepsis. I know of no field of medicine that is so fascinating and I know no group of men from whom I have learned so much as from the otologists. From the earliest days I have been privileged to work with men who have been especially interested in sepsis. It strikes me that the main function of the medical man is to work hand in hand with the otologist. It so often happens that we see the cases after the complications have developed, and we have to attempt to determine which came first, whether the focus distant from the ear followed the ear infection, followed the operation or was present before the operation. It is an extremely important point to determine whether or not the sinus thrombosis is operable in a patient with a metastatic focus.

I might quote a case in point. Following a mastoid operation the patient developed pain in the sternoclavicular joint. The temperature subsided to a lower point, with only a mod-

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erate elevation of the leukocytes. The blood culture was negative. The area of pain developed into a mass. Aspiration of the mass revealed pus. The organism was hemolytic streptococcus. Had this patient an active sinus thrombosis, or had this patient had a general blood stream infection at the time of operation and was this a secondary metastatic focus at that time? The medical opinion was that had there been a sinus thrombosis it would have taken care of itself, and all that was necessary was to incise the focus and watch. The patient recovered perfectly without a sinus operation. However, the problem is much more complicated. All cases are not that smooth. The secondary focus often becomes manifest a long period of time after the primary mastoid operation, the blood culture becomes positive and the question up to the medical man is whether this is or is not a sinus thrombosis. The presumptive evidence is of course in favor of sinus thrombosis, but there are cases in which the secondary focus in itself may become the primary focus. That is especially true in osteomyelitis cases. The sinus thrombosis may have completely healed and a suppurative focus in the bone may in turn give a positive blood culture. The sinus is exposed and nothing is found. To determine whether the secondary focus has in itself become a primary focus and is feeding into the blood stream again is a rather important point.

As far as specific methods of treatment directed toward general sepsis is concerned, the problem in otology is very little different from the problems in general sepsis in any other part of the body. We have no specific method of therapy. There are no specific chemicals which are powerful enough to kill the organisms in the individual without in all probability destroying the individual. We have experimented with dyes, acriflavine, etc., very extensively. I will never use them again. These chemicals have not been beneficial, but harmful. I thoroughly appreciate the records in the literature, especially those from Hopkins, that injections of acriflavine have been beneficial. These records after careful review have not been conclusive. The series is too small. We find in the experience of all of us that cases of general sepsis sometimes recover spontaneously. I rather feel that most of the records in that group are related to spontaneous recoveries rather than to the dye therapy. In fact, in my own experience, dye therapy is extremely harmful.

Another point to bring up is the value of transfusions in general infections. I feel that transfusions have been greatly overdone. As soon as it has been determined that the patient has a generalized infection, the frequent attitude is to transfuse in the hope that transfusions will do something which we do not quite understand. We hear it spoken of as stimulation of the myeloid elements, stimulation of the bone marrow. I feel that the indications for transfusion are rather definite. Of course, the commonest indication is a rapidly declining hemoglobin due to a hemophilic organism. Rather small transfusions repeatedly given are best in order to attempt to compensate for the loss of blood. It is also definitely indicated of course where there is an actual severe anemia. My own rule is, when the hemoglobin is 60 or below, always transfuse. When it is above 60, hold off. I have not been able to convince myself that transfusions are serviceable in controlling infections. The only method of controlling the infection is, I believe, to eradicate the primary focus. The transfusion can then be used as a supportive measure. As far as using the blood of immunized donors is concerned, the remarks previously made apply.

Dr. Dixon summarized nearly all I wanted to say about therapy in two words. He said that the fundamental treatment is to support the patient and force fluids. I think that is true. I am rather partial to forcing fluids as far as one possibly can. If the patient cannot take it by mouth, use all other methods. If possible, give fluids by rectum. If the rectum is too irritable and sufficient fluid cannot be forced that way, the slow intravenous drip of glucose and saline will be a very satisfactory method. One can get in anywhere from five to seven liters in 24 to 36 hours.

It must be remembered that infection of the mastoid is rarely a primary disease, but is secondary to some other infection, such as influenza, measles, scarlet fever, pneumonia, etc. This is an important point in determining whether the associated pathological condition is an accompaniment of the primary disease or a complication of the otitic infection. This is well exemplified in pneumonia. We see cases of pneumonic consolidation in individuals with otitic infection. Is the consolidation the result of the otitic infection or part of the primary disease from which the patient is suffering? If the blood culture reveals a pneumococcus Type 1 or 2, one rarely

if ever finds a true sinus thrombosis. In these cases the use of the specific sera may be of value. With pneumococcus Type 3, where one gets a peculiar type of sinus thrombosis a true thrombophlebitis of the sigmoid sinus will follow. At times erysipelas develops around about the ear previous to or following operation. Here the erysipelas serum may be of value.

In the scarlet fever cases the problem is very different and difficult, especially in those cases of scarlet fever with labyrinthitis. That problem has not been brought up here tonight, and I do not want to touch upon it, but I have seen three cases in the last two years of acute suppurative labyrinthitis following scarlet fever. Of the otologists in town, some advise operating and some advise leaving alone. The one case I saw recover had had nothing done. The child was simply left with the deafness and subsequently a mastoid operation was performed.

The diagnostic problems associated with otitic infections are tremendous, and one of the commonest ones we meet is abdominal pain. A prominent pediatrician in this town said if he died he would like them to write on his tombstone that he died from a belly-ache, but not his own. I think that is true and especially in otitic infections in children. We see so many of them with abdominal pain. The problem comes up, is this part of a general infection or are you dealing with a suppurative process in the abdomen? It is surprising how often an acute gangrenous appendicitis is found as a complication. Only recently I saw one. We see it in infections of the upper respiratory tract and with ear infections. Very often the abdominal pain is only part of the general infection. It is an exceedingly difficult problem to determine and requires very careful observation and watching. I have seen only recently a patient who was operated on for a suppurative condition of the tonsils. The patient had abdominal pain which was totally ignored. Four days later the patient was operated on for gangrenous appendicitis with an abdomen full of pus, because the abdominal pain had been considered only part of the ear infection. This is not an uncommon fact.

I might quote a case I saw only recently that was even a greater diagnostic problem. The patient had had an otitic infection, a positive blood culture; the sinus was operated

and the jugular vein not ligated. The temperature persisted and the anemia persisted, and no cause was found. Then the jugular vein was ligated. There was no change in the clinical picture. Then the patient began to complain of pain in the lumbar region. The diagnosis was then suspected, X-rays taken and there was found to be a perinephritic abscess which had occurred previously, probably at the time of the sinus thrombosis when the blood culture had been positive. The drainage of this perinephritic abscess brought about complete recovery of the patient. This is very similar to the first case presented this evening, where a suppurative condition of the hip joint was found after the patient had been subjected to an operation or two.

I feel that you are all alert and anxious to get at this controversy whether the jugular should or should not be ligated. I would like to say that it is an education, I think, for the medical man to follow a case from the beginning. It gives him a much better idea how to advise, instead of being called in afterward, not knowing the entire problem. It is difficult for the medical man to evaluate in time the remote problems from the ear. The treatment of sepsis consists of transfusions, proper food, forced fluids. In seeing these cases of otitic infection early, the medical man can in the course of time possibly learn more about infections and apply it to infections in other portions of the body.

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THE PATHOLOGY OF THE SPREAD OF OSTEOMYELITIS OF THE SKULL.*

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There occurs perhaps no more tragic sequel to nasal accessory sinus disease than that of osteomyelitis of the cranial bones. Its ravaging effects upon the calvarium, its devastating attack upon the meninges and brain, its ofttimes slow, progressive invasion of most of the osseous tissues of the head with gaping wounds and hideous deformities make it a tragedy hardly comparable to any other in the field of surgery. When with this picture before us, there is reason to suspect that perhaps some error of omission or commission on the part of the surgeon is responsible for these grotesque changes, the tragedy is complete and the true significance of this grave complication is clearly understood.

We view with alarm the frequent occurrences of osteomyelitis of the cranial bones, following operations upon the nasal accessory sinuses. Many such reports are found in the medical literature and numerous cases undoubtedly pass by without being recorded in our scientific journals. Most laryngologists with whom this problem is discussed have had at least one personal experience with diffuse infection of the calvarium, and its common occurrence following surgical procedures upon the paranasal sinuses gives rise to the belief that the association is one of cause and effect and not a matter of mere coincidence.

My individual experience in this connection has furnished some interesting observations. During the past 18 years I have had the opportunity of studying 26 cases of diffuse osteomyelitis of the cranial bones. In all but six cases the calvarial infection followed an operation upon one or more of the nasal accessory sinuses. In most instances the Killian frontal sinus and ethmoid operation preceded the diffuse infection of the skull, though the intranasal procedures were

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also apparent forerunners of this grave complication. In one patient an attempt to enlarge the frontonasal duct was followed by a widespread infection of the cranial bones; in another a simple intranasal drainage of the antrum appeared to initiate the attack; while in two more cases the Caldwell-Luc technique upon the maxillary sinus was followed by a devastating invasion of the calvarium. Thus it may be shown in my own series of cases and confirmed by numerous reports in the medical literature that osteomyelitis of the skull may and has followed most of the surgical procedures upon the nasal accessory sinuses; the intranasal as well as the more radical external operations.

This serious complication is not limited to the experiences of professional men of meager training. It has occurred in the hands of master surgeons as well as in those of less surgical skill. No one of our profession is immune to this grave accident, and none has offered a precise explanation of the influences which contribute to the development of cranial osteomyelitis, following well directed surgical efforts upon the nasal accessory sinuses.

In analyzing many of the records appearing in the medical literature one is impressed with the fact that in many instances inadequate drainage of the sinus followed the primary operation and that a circumscribed collection of pus under pressure was allowed to remain within the sinus and neighboring soft tissues until extension to the adjacent bones of the skull was in evidence. Frequently under these conditions the surgeon is prone to temporize in the belief and hope that the process will limit itself and resolve in the course of a few days, and that further interference with a patient who has already passed through a major operation will not be required. The gravity of this complication must be clearly recognized. It possesses dangerous potencies and in my opinion calls for an immediate secondary operation of such a character as to lay widely open the area of infection, that adequate drainage may be promptly effected and maintained.

Our interest is directed to a study of the pathways along which infection may travel from the nasal sinuses to the cranial bones. Obviously the first to attract our attention is the spread of inflammation by continuity of tissue. This mode of diffusion seems the logical one for calvarial infection aris-

ing from a suppurative process within the frontal sinuses, since the latter are virtually eroded diploe in more or less intimate relation to the diploic structure of the frontal bone. Here we observe the direct extension of infection to the frontal diploic venous system which lies between the outer and inner tables of the skull, producing a septic thrombophlebitis and the typical osseous changes of an osteomyelitis of the cranial bones.

The frontal diploic veins as seen in radiographs of dried skulls tend to converge downward and forward, and to find an exit from the frontal bone through a small efferent channel in the superciliary ridge. To me this presents an important practical application in that it affords exposure of the frontal diploic venous system to suppurative processes arising within the soft tissues in and above the orbit following radical external operations upon the frontal and ethmoidal sinuses where adequate drainage has not been secured. The knowledge of this anatomical fact further emphasizes the urgent need of prompt secondary drainage when purulent collections arise in close proximity to the supraorbital ridge.

The diffusion of infection by continuity of tissue, however, does not complete the pathological picture. It does not account for the development of separate patches of osteomyelitis in the calvarium; it fails to offer an acceptable explanation for the extension of a suppurative process in the maxillary sinus to the cranial vault, and it cannot explain the presence of a variety of pathological changes which I will endeavor to describe as they were observed in postmortem studies of osteomyelitic lesions of the cranial bones.

A study of calvarial infections is prone to impress the student with at least one significant observation, namely, the pathology of the spread of infection along the surface of the dura beneath the inner table of the skull. Here we observe an exudate of variable thickness and in different stages of organization extending along the dura, exerting its destructive influences either through hematogenous metastasis or by pressure erosion. It has been a constant finding in all of my cases when observed during operation or examined postmortem. It has, I believe, an exceedingly important clinical significance and must be given due recognition in the selection of thera-

peutic measures to combat this dreaded disease and its serious intracranial complications.

That we may more clearly understand the influences and behavior of these pathological changes upon the dura, let us turn to a brief consideration of the normal vascularization of the bones of the calvarium. It will be observed in radiographs of the skull, following injection of the external and internal carotid arteries with a radiopaque substance, that the great bulk of the blood supply to the cranial vault comes to it from the inside of the calvarium. The meningeal arteries freely vascularize the dura mater and send a dense network of nutrient twigs through the internal table into the flat bones of the skull. The dura, particularly its outer layer, is virtually the internal periosteum of the skull. The external periosteum is much less vital to the nourishment of the calvarium, for it can be demonstrated that it furnishes but a meager supply of blood through fine arterial twigs distributed chiefly over the temporal and occipital regions.

The diploic veins of the cranial vault are often clearly visualized in routine Roentgenograms of the skull, particularly in patients in the later years of life. They lie within the substance of the bone and occur in three main groups, the frontal, parietal and occipital systems, occupying the corresponding bones of the head. From these veins a few efferent vessels arise which penetrate the external table of the vault and communicate externally with the veins of the soft tissues of the scalp. It is to be noted, however, that the great bulk of venous drainage from the diploic systems is through larger efferent channels which pierce the inner plate of the skull and empty into the meningeal vessels and the large intracranial sinuses. Thus we observe as in the case of the arterial supply to the cranial bones that the venous drainage is chiefly within the vault of the skull.

We therefore recognize the important physiological relationship between the dura, with its free vascularization, and the overlying cranial bones, while viewing with speculative interest the destructive effects which may arise from pathological processes developing within the potential space between the dura and the cranial vault.

That the veins of the mucous membrane lining of the frontal and ethmoidal sinuses communicate directly with the dural

vessels is not difficult of proof. It can be demonstrated further that these veins may participate in a septic thrombophlebitis which tends to distribute infection to the various bones of the skull. The typical pathological formation is that of a septic exudate spreading over the surface of the dura and occasionally extending far beyond the original site of infection. Within the exudate various stages of organization are observed. It contains a varying amount of fibrous connective tissue and everywhere a high degree of vascularity with evidence of a fulminating perivascular extension of infection in all directions.

The influences of such an inflammatory exudate are twofold. *1.* It produces a thrombophlebitis of the efferent diploic channels which drain the diploic venous system into the meningeal vessels and through retrograde thrombosis, subsequently carries the infection to the diploi of the skull. *2.* It separates the dura from the cranial bones and tends to cut off the chief blood supply of the calvarium, thereby depriving it of adequate nutrition. The combined effects of these influences, to which the cranial bones in osteomyelitis are often exposed, tend to nutritionally impoverish the osseous tissues and lay them open to a widespread infection.

In support of the view that osteomyelitis of the calvarium may start its invasion on the under surface of the cranium in the pathological changes above described, the following studies are enumerated:

1. It has been demonstrated postmortem that this mode of diffusion may be responsible for the occurrence of isolated foci of osteomyelitis in the cranial vault. Here we may find an osteomyelitic lesion somewhat removed from the primary infection and separated from the latter by a zone of healthy bone.

2. Postmortem examinations not infrequently reveal septic thrombi within the efferent channels of the diploic veins and an exudate spreading over the dura in advance of bone suppuration. These processes may be found extending beneath the inner table of the calvarium ahead of the inflammatory disease in the overlying osseous tissue. The exudate may show a fibrous connective tissue organization of several days' duration, though the part of the cranial vault immediately over

this process may be observed under the microscope to be free from an osteomyelitis. This has been a constant finding in all of the autopsy material which has come to my attention. It appears to furnish satisfactory proof that the changes upon the dura are primary and not a secondary development in the course of an inflammatory disease of the cranial bones.

3. Pathological studies of calvarial infections furnish innumerable signs of early injury to the inner table of the skull. In my experience the inner plate of the infected calvarium has been the first to be destroyed. But what is of still greater significance is the fact that the inner table may be the only part of the bone primarily affected while the overlying medullary tissue and external plate may not become involved. In this alone we have considerable proof that the primary invasion and subsequent spread of infection is between the dura and the inner table of the skull.

The problem of treatment in osteomyelitis of the cranial bones is solved through an understanding of the pathological phenomenon above described. Obviously there is but one way to adequately drain and eradicate the disease, and that is by the radical removal of full thickness of all of the bone which is the seat of infection. By this means alone can we dispose of the carious and necrotic bone and secure mass drainage of the products of inflammation which are scattered over the meninges. It is moreover essential to keep the scalp separated from the dura by packing until resolution of the infection has occurred. Failure to observe this detail may lead to a dense union of scalp and dura with the formation of isolated pockets of pus. A collection of this kind may terminate in meningitis or brain abscess, though all of the diseased bone may have been completely removed.

In a previous publication, I have discussed more precisely the surgical treatment of calvarial infections. I have pointed out that the disease is a highly destructive one and calls for surgical interference of a formidable character. It has been said that the removal of the external table and medullary structure of the cranial bones will often suffice to render a cure. This measure has been suggested in order that the inner table may be preserved as an osteogenetic medium for the regeneration of osseous tissue. To this method I cannot subscribe. The extent and location of the pathological changes

in this disease require the excision of the entire thickness of infected bone.

There need be no apprehension in resorting to such radical measures, since the regeneration of bone usually occurs in skull defects of this character to an amazing degree. I have shown that embryonic bone may arise from any fibrous connective tissue background, including the dura, and that the amount of regeneration usually suffices to close the perforation and to closely restore the contour of the cranial vault to normal. This has occurred without exception in every patient in my series where recovery followed the removal of small or large portions of the cranial vault.

CONCLUSIONS.

1. Diffuse osteomyelitis of the skull follows with alarming frequency operations upon the nasal accessory sinuses. We are at a loss to account for this grave complication, though in some cases it has probably developed as a result of inadequate drainage and the neglect of purulent collections about the soft tissues of the orbit and frontal bone. A word of caution is voiced against delay in securing wide secondary drainage when such accumulations are formed.

2. Many cases of calvarial osteomyelitis are chargeable to an intracranial, yet extradural, infection which spreads between the dura and the inner table of the cranial bones. We find in autopsy material abundant evidences of this mode of extension of inflammation in osseous tissue.

3. Treatment must be of a radical nature. It calls for the removal of full thickness of all of the diseased bone and the maintenance of massive drainage of the exposed dura.

4. The regeneration of bone to an extraordinary degree is the usual result. By this phenomenon enormous calvarial defects are closed with restoration of the cranial vault to a nearly normal contour.

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LATENT OSTEOMYELITIS OF THE SPHENOID BONE REACTIVATED BY TRAUMA WITH DEATH FROM MENINGITIS.

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A case of latent osteomyelitis of the sphenoid bone reactivated by trauma with death from meningitis is presented in order to illustrate the importance of careful examination and early recognition of sphenoid suppuration.

There are two types of osteomyelitis resulting from nasal accessory sinus suppuration, spontaneous and postoperative. There may be a combination of both, in which a latent chronic spontaneous osteomyelitis is reactivated following postoperative trauma.

Before reviewing the literature on the subject of osteomyelitis of the sphenoid bone or referring to the anatomy, I wish to present a brief history of a case recently under observation.

Case Report: Female, white, age 18 years, had been treated for some time for chronic maxillary sinusitis and ethmoiditis. Her first appearance in the clinic was in July, 1926. At that time she complained of difficulty in breathing and considerable thick nasal discharge. Examination revealed swollen, boggy, nasal mucous membrane. No other observations or diagnosis were made at the time.

She was seen about three years later, in August, 1929, for an acute otitis media. No other notes or references were made as to her nasal condition.

In October, 1930, the patient entered again. This time the patient complained of headaches and nasal discharge. Summary of her present history was as follows:

Present Illness: Patient complained of severe morning frontal headaches of four years' duration, becoming progres-

*Deceased.

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sively worse. These headaches radiated to the occiput and ameliorated toward the afternoon. Patient hawked considerably, especially in the morning, and complained of profuse nasal discharge. No other subjective symptoms elicited.

Past History: Tonsillectomy and adenoidectomy four years ago; diphtheria at three years of age. There is no history of other illness or operations.

Family History: Essentially negative.

Physical Examination: Examination of the throat revealed tonsil tabs in both tonsillar fossae, with thick tenacious foul, yellow green purulent discharge on the postpharyngeal wall. Teeth were essentially negative. There was considerable discharge in the middle and inferior meati of the left nares, causing as a result partial obstruction. The character of the secretion was the same as in the throat. The nasal secretion was removed with suction. Examination revealed small polyps and polypoid degeneration in the region of the anterior part of the middle turbinate on the same side. The right area was essentially negative. There was marked left supraorbital tenderness.

Transillumination revealed markedly diminished radiance of the entire left side of face.

Patient was seen five days later. The polyps were removed and an anterior ethmoidectomy was performed under local anesthesia. Two days later the patient returned to the clinic. Examination of the nose revealed an abundant thick yellow purulent discharge in the region of the operative field.

The X-ray report showed pathology in the left frontal sinus and ethmoid with thick membranes in both antra.

Urine and Wassermann were negative. Patient was referred to the house physician with a diagnosis of left pansinusitis.

Summary of House Record: Patient entered the hospital on Nov. 21, 1930. The following day a left radical maxillary operation was performed with a partial transantral ethmoidectomy. The lining mucous membrane of the antrum was found hypertrophied and polypoid. There was a profuse amount of free pus which was removed with suction. The lining polypoid membrane was removed intact. The antronasal wall was perforated with trochar and the opening was

enlarged. Two iodoform drains were inserted. The gingivolabial incision was closed with two chromic catgut sutures. The postoperative course was uneventful. Packs were removed 48 hours following operation. Sutures were removed two days following this. The wound in the mouth was clean. There was a slight amount of yellowish discharge in the left middle meatus. Eight days following the operation the patient began to hemorrhage profusely from the left nares. Considerable effort was made to arrest the hemorrhage, requiring nasal packs and postnasal sponges. The following day the temperature began to rise. The patient complained of generalized stiffness of all joints. Extension and flexion of the head caused some pain. The packs were removed and temperature dropped to normal the same day. The patient became progressively worse. There was a suggestive Kernig, the knee and ankle jerks were not obtained, and there was no Babinski present. The fundus examination showed nothing abnormal. Stiffness of neck became more pronounced.

The blood picture was essentially negative except for a white count of 23,000, with a high percentage of polymorphonuclears.

A neurological consultation on Dec. 5, four days following the onset of the present condition, confirmed the diagnosis of meningitis. A lumbar puncture was performed, revealing a cloudy fluid under pressure of 580 mm. Dynamics were normal. There was a prompt rise of individual and combined jugular compression. The examination of the spinal fluid revealed a strong positive Pandy reaction. Qualitative tests showed no sugar. Smear of the centrifuge sediment showed many white cells but no organisms. A count of the fluid showed 9000 cells, all of them polymorphonuclears.

The patient was seen about 20 minutes following a lumbar puncture, at which time she was quite comfortable and breathing quietly. About one-half hour after this she was again seen by the house officer. The patient appeared quite pale on close examination and pulseless. She was pronounced dead about one hour following the lumbar puncture.

Autopsy: At autopsy a thick exudate covered the pons and extended over the inferior surface of the cerebellum. There was a small pressure cone present. This is often seen in

patients who have not died suddenly, according to the observations of the neurological consultant. Dissection of the brain revealed no abscesses, but there was a slight dilatation of both ventricles somewhat more marked on the right. The fluid in the right lateral ventricle appeared to be somewhat purulent. Stripping the dura from the anterior fossa revealed no abnormalities. The ethmoids on both sides and the sphenoidal sinus were full of thick pus; that in the sphenoidal sinus was much thickened and dirty brownish gray in color. On raising the dura in the region of the left Gasserion ganglion, a reddish brown purulent material was seen apparently between it and the base of the dorsum sellae on the left. Stripping the dura from the dorsum sellae its anterior surface showed irregular thickening, evidently chronic osteomyelitis. This process extended through the dorsum and was continuous with the pus in the sphenoidal sinus.

Middle ears and mastoids: Negative.

Anatomical Diagnosis: Meningitis, sinusitis, and osteomyelitis of skull.

Microscopic Examination: Brain: Meninges thickened by fibrin enmeshing polymorphonuclears with hemorrhage and necrosis. Large areas of necrosis with polymorphonuclears and fibrin. Pituitary: Negative. Choroid plexus: Negative.

A number of similar cases have been reported in the literature, a few of which are recorded here. Blumenthal[1] reported a case involving the greater wing of the sphenoid of a child, age four years. Flatau[2] reported a case of caries of the sphenoidal sinus in a young girl with bilateral sphenoidal sinus suppuration. Her chief complaint was headaches. Both sinuses were opened with no relief. She was admitted to the hospital in a somnolent condition and died a day later. Autopsy revealed meningitis caused by caries of the sphenoid bone with spontaneous perforation into the cranium. Lench[3] reported a similar condition, in which a patient, age 25 years, with latent chronic sphenoiditis, according to history, took sick with fever and headaches following a blow on the head. The inciting organism was reactivated in the sphenoid bone, resulting in an acute osteomyelitis with thrombosis and meningitis. Patient died in 18 days following onset of condition. Autopsy revealed basal meningitis, abscess of the right frontal

lobe, suppurative thrombosis of the cavernous sinus of the right sphenoid, of the right petrosal, osteomyelitis of the sphenoid bone and occipitobasilar portion. Hoston reported a case of thrombosis of the cavernous sinus resulting from inflammation of the sphenoid sinus associated with osteomyelitis necrosis and gangrene of the basilar sphenoid and occipital region.

Osteomyelitis of the bones of the skull occurs more frequently than is generally thought. However, in comparison with the same pathological process occurring in long bones, it is rather rare. The involvement of the latter being six to eight times more frequent than those of the flat and short bones of the skull and wrist.

Most of the osteomyelitis of the flat and short bones involve the upper jaw and quite commonly are the result of severe sinus infection. Generally, suppuration of the sinuses involve the mucosa alone, and rarely the bone. Why osteomyelitis develops in one patient with sinus infection and not in others under conditions which may be apparently the same is difficult to explain except to say that constitutional resistance and the virulency of the infecting organism may play some part in the process.

The more frequent occurrence of the osteomyelitis of the long bones may be due to various factors. Trauma plays an important part in this disease, more frequently involving the long bones of the upper and lower extremities than the flat and short bones of the skull and wrist. Another important factor is the histological structure and blood supply of the two types of bone.

The nutrition of bone is through the periosteum and by special nutritive branches through the foramina. Although the blood vessels to the flat and short bones are not as large as those which supply the long bones, they are more numerous.

Histologically,[4] bone consists of modified connective tissue presenting certain structural peculiarities in different bones and in different parts of the same bones. It is covered by periosteum which has a rich blood supply. Adjacent to the periosteum is the compact portion of the bone and still deeper

the spongy layer, the quality of each depending on the type of bone and may not be at all uniform in parts of the same bone.

Long bones have a thick, compact layer in comparison to the flat, short bones, which have a thin, compact portion. The spongy layer is present to a greater extent in the flat, short bone, in the meshes of which is the bone marrow.

In these flat and short bones is found marrow. This type of marrow consists of connective tissue reticulum with cellular elements as multinucleated and uninucleated giant cells, leukocytes in various degree of development. Fat varies here, but is not abundant.

This histological difference accounts for the less frequent occurrence of osteomyelitis of the flat and short bones in comparison to long bones.

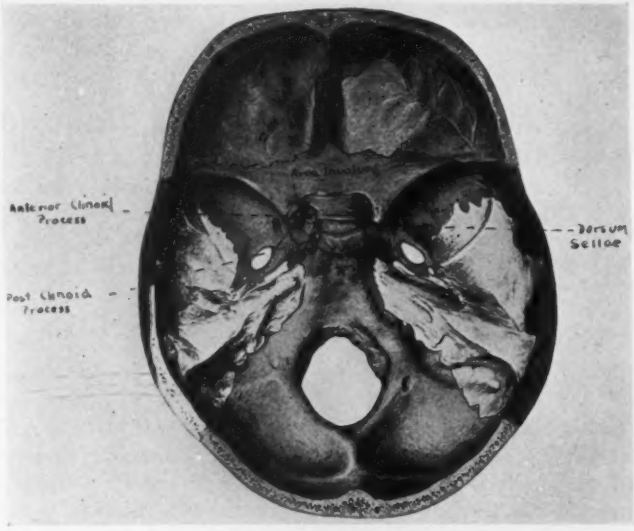
The symptoms present in suppuration of the sphenoid sinus may be vague or common to pus in any of the accessory sinuses. The most frequent complaint is postnasal catarrh and pain between and behind the eyes radiating to both temporal regions and the occiput. Eagleton[5] regards pain in midline on top of the head as diagnostic of sphenoid suppuration. Eye symptoms which may be present are lachrymation photophobia, blepharospasm, and transitory scotoma. Anosmia of cacosmia may be present.

Examination of the nose will reveal at times various types of secretions in the regions common to suppuration of the posterior series of sinuses. Besides the local symptoms which may be present, one may encounter constitutional reactions. Fever is present in the acute condition, may be present but is usually absent in the chronic suppurative processes. Symptoms referable to disturbances in the respiratory or digestive tract may also be present.

The complications resulting from an osteomyelitic process involving the sphenoid bone are few but almost always disastrous and almost always the result of perforation through the walls of the sinus. The most frequent is a basal meningitis and cavernous sinus thrombosis. Other complications which may occur not too infrequently are phlebitis of the cavernous sinus with septicemia, abscess of the brain, sup-

purative encephalitis and intracranial hemorrhage. This can be readily understood when reviewing the anatomical relations in this region.

The sphenoid bone[6] is situated in front of the basilar portion of the occiput at the base of the skull. It is composed of a body or median portion and two great and small wings extending laterally from the body and pterygoid processes projecting from below. The sphenoid sinuses are two large irregular, assymetrical cavities separated by a septum. It is hol-



Anterior Superior View of the Sphenoid Bone.

lowed out of the interior of the body of the sphenoid. They are present at birth and attain their full size anywhere between the age of six and puberty.

The upper surface of the body, which is the delicate roof, is in relation to the cribriform plate, olfactory lobes of the brain, optic chiasma and more posteriorly to the hypothysis cerebri. It is through this portion of the bone that erosion takes place. Posteriorly to the fossa is the dorsum sellae, on

either side of which there is a notch for the transmission of the abducent nerve. Below this notch is a process articulating with the apex of the petrous portion of the temporal bone. The superior angles of the dorsum sellae end in two tubercles, the posterior clinoid processes. The optic foramina which is formed by the two roots of the small wing as it unites with the body of the sphenoid transmits the optic nerve and the ophthalmic artery.

Laterally the body of the sphenoid is attached to the great wings and the pterygoid processes below. There is a broad groove above the attachment of each wing lodging the cavernous sinus and the internal carotid artery. Both are in close relation to the sphenoidal sinuses and extension by contiguity or through thrombotic vessels takes place very easily.

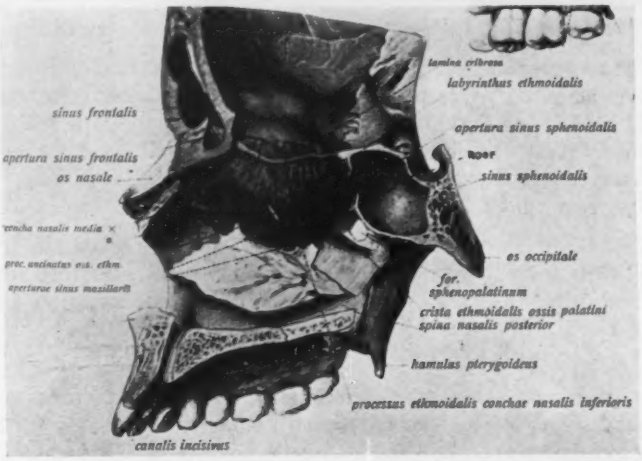
Posteriorly, the body is in relation to the basilar part of the occipital bone on which lodges the pons. Erosion takes place here quite commonly. Anteriorly the body presents a crest which articulates with the perpendicular plate of the ethmoid, forming part of the nasal septum, on each side of which there is an irregular opening into the corresponding air sinus. The lower front portion is closed by two curved plates of bone, the sphenoidal conchae.

It is interesting to note the relation of the small wings to the body of the sphenoid and consequently to any diseased process in the air sinus. The small wings are two triangular plates arising from the upper and anterior parts of the body and project laterally. The medial end of the upper surface supports the frontal lobe of the brain, the inferior surface goes into the formation of the back portion of the roof of the orbit and upper boundary of superior orbital fissure. This fissure connects the cranium with that of the orbit and transmits the third, fourth and fifth cranial nerves, together with branches of the ophthalmic, some filaments form the middle meningeal and recurrent branches from the lachrimal artery and the ophthalmic vein.

One may easily conceive how a suppurative process involving the sphenoid sinus may produce fatal results, especially through defects, dehiscences or venous channels between the bony wall, the adjoining dura and cavernous sinus. In adults,

with united sutures these venous channels communicate freely with one another. They are composed of numerous network of veins which are large and contain pouch-like dilatations serving as a reservoir.

The pathological process is essentially the same as in long bones. In the spontaneeous type, the mechanism of extension depends on several factors, as virulence of the organism, resistance of the patient, and on the character of the involved bony anatomic structures. The appearance of the pathological



Sagittal section of skull, showing relation of sphenoid sinus to nose, throat and cranial fossa.

process varies with the stage of the disease. The diploe may on gross appearance show beginning areas of congestion. Associated distention and thrombosis takes place as the result of leukocytic invasion with necrosis and liquefaction of the cellular substance. Absorption of bony tissue with the formation of multiple abscesses produces widespread destruction of tables of the skull. These abscesses sometimes coalesce and extend along the marrow. The process produces a separation of the overlying periosteum, cutting off the line of nutrition. Bone proliferation and sequestrum formation re-

sults as the infection is prolonged. Systemic dissemination of the bacteria with resulting septicemia and pyemia may then occur. Although attempts of bone regeneration may be present, the destructive process predominates throughout, the neighboring organs becoming involved and death from intracranial complications ensues. In some instances the infective process may remain limited and quiescent indefinitely. However, injury with impaired resistance leads to a reactivation of the process.

According to literature the usual bacteria described are staphylococcus aureus, streptococcus, and pneumococcus. Frequently one may see contamination with other organisms which normally are found in the upper respiratory tract.

According to postmortem examinations, sinus disease is present in 30 per cent of all subjects. The most commonly affected is the maxillary and the sphenoidal, the latter being more frequently involved than is suspected, as Lermoyez observed: "Sphenoidal suppuration is not rare; it is only its diagnosis which is uncommon."

St. Clair Thomson[7] reported a series of cases of meningitis with cavernous sinus thrombosis following long standing suppuration of the sphenoid in which the cerebral invasion was not due to trauma or to suppuration of any of the other accessory sinuses.

Treatment: One should become suspicious of troublesome postnasal catarrh, headaches and ocular disturbances. The diagnosis once established, this condition is amenable to proper treatment. The treatment varies from the ordinary cleansing measures to more complicated surgical interferences. This may consist of enlarging the opening of the sphenoid sinus or the removal of its anterior wall preceded usually by a middle turbinectomy and ethmoidectomy. The results here are certainly more encouraging than in treatment of suppuration of the other sinuses when performed by competent and well trained rhinologists.

SUMMARY.

A case of sphenoid suppuration resulting in osteomyelitis and death from meningitis is described. The object of this description is to call attention to the wisdom of early recog-

nition of sphenoid suppuration and the careful observation of such cases.

The danger of osteomyelitis and meningitis due to anatomical and histological structure is stressed. Special reference is made to the frequency of erosion in region of delicate roof of bone; and extension along thrombotic vessels to neighboring structures as described.

A complete X-ray study with careful clinical observation would serve to avoid such serious consequences.

REFERENCES.

1. BLUMENTHAL, B.: Osteomyelitis of Greater Wing of Sphenoid. *Ztschr. F. Laryngol. u. Rhinol.*, 1928-1929.

2. FLATAU: Caries der Keibeinhöhle. *Berliner Klinische Wochenschrift*, Oct., 1895.

3. LENCH, O.: Osteomyelitis acuta des Sphenoidale. Thesis Univ. of Basel, 1930.

4. PEIRSOL: Histology.

5. EAGLETON.

6. Gray's Anatomy.

7. THOMSON, ST. CLAIR: Cerebral and Ophthalmic Complications in Sphenoidal Sinuses. *Lancet*, 1913.

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RECENT EXPERIMENTAL WORK ON PHYSIOLOGY OF HEARING. ITS SIGNIFICANCE TO THE OTOLOGIST.*†

DR. M. H. LURIE, Boston.

In 1930, Wever and Bray of Princeton, reported that by placing an electrode on the eighth nerve of a cat and connecting this electrode with an amplifying apparatus of the radio type, they were able to hear in the loud speaker of the radio an exact reproduction of sounds, noises and voices that were sent into the cat's ear. This discovery is known as the Wever and Bray Phenomenon. As a result, there was opened a new field for research work to be done on the physiology of hearing. This important discovery immediately provided, for those of us interested in the function of the cochlea and the middle ear, a method of working with the living mechanism. Previous to this, practically all the experimental work done was limited to reconstructing models and devising mechanical cochlea.

This recent research has given us enough evidence to make the following predictions: *1.* That there will be a revival of middle ear surgery for the relief of certain types of deafness; *2.* That the theories and physiology of hearing will be placed on a firm or even permanent basis; *3.* That direct experimental investigation can be done on the auditory pathways and other nerve pathways in the brain.

The work was taken up at about the same time at Johns Hopkins Medical School under Dr. Crowe and Dr. Hughson, and at the Harvard Medical School under Professor Davis and Dr. Saul. It soon became evident that the viewpoint of the otologist would be of help and the speaker became a member of the team.

In Dr. Crowe's laboratory at Johns Hopkins University, the work centered chiefly around the function of the drum, ossicles and muscles of the middle ear. The Wever and Bray

*Read at the College of Physicians, Philadelphia, Pa., Nov. 15, 1933.
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phenomenon was taken as a criterion of the animal's hearing. The work done on the function of the middle ear apparatus was based on the results obtained through placing a broad electrode in the brain, in or near the eighth nerve, and picking up the response from that region.

At the Harvard Medical School the interest was chiefly in the analysis of the phenomenon reported by Wever and Bray in determining what happens in the cochlea and tracing the auditory nerve impulses up through the auditory tracts to the cortex of the brain. The work has been practically continuous from 1930 to the present time. Considerable investigation has been done and a number of reports have been made. The time has now come to consider not only the progress that has been made, but also to ascertain what clinical value it may have to the otologist at present or in the near future.

The first problem attacked was an analysis of the phenomenon. It soon became evident that the Wever and Bray effect was composed of two components, one arising in the cochlea itself, and the other being true nerve impulses coming up the cochlear nerve to the brain. These nerve impulses, which may be detected by the electrical disturbance or "action currents" associated with them, were studied very carefully and traced through the brain stem from the eighth nerve. The various ganglion stations and tracts between the geniculate body and the cochlear nerve were studied; that is the inferior colliculus, the lateral lemniscus, the stria acustica, trapezoid body and the cochlear nuclei. It was found in this work on the nerve impulses that sounds sent into the cat's ear up to 1000 vibrations per second were reproduced by the nerve impulses and they were traced from the eighth nerve up as far as the medial geniculate body. Later with improvement in apparatus and technique, we were able to detect, faintly, nerve impulses synchronous with the sound wave up to about 2800 vibrations per second. The study of the function of the primary cochlear nucleus and the nuclei higher in the brain stem proved that the sound was not of as pure a type as that heard from the eighth nerve. There seemed to be a blurring of the sound as if many additional nerve impulses traveled up the auditory tract along with the synchronous impulses, thus modifying the response received back in the loud speaker of the apparatus. The nearer the tone sent into the cat's ear

approached 2800, the greater the proportion of nonsynchronous nerve impulses. Above 2800 we have been able to detect no tone in the auditory tracts, but simply a rushing noise when these high tones were sent into the cat's ear. In other words, the impulses traveling up the auditory tract were no longer synchronous with the vibrations making up the sound. This probably indicates that the cochlea itself analyzed the sound sent in above, and perhaps also below, 2800. As the nerve impulses pass from a lower nucleus to a higher nucleus they became more and more nonsynchronous. Above the medial geniculate body they are sent to the auditory area of the cortex simply as nerve impulses and the interpretation of the sound heard is probably done in the cortex, possibly due to specific stimulation of certain parts of this area. Along with this work considerable study was given to the interval of time it takes a nerve impulse to travel from the cochlea to the cochlear nucleus, to the nuclei in the lateral lemniscus, inferior colliculus and medial geniculate ganglion.

A study was also made of the phenomenon occurring in the cochlea itself. The cochlear response, which resulted from a sound wave entering into the membranous cochlea, could be detected if the sound was loud enough and the amplification great enough, practically all over the skull and brain of the cat. The two best points at which to study this component were found to be the round window with its surrounding bone in the middle ear, and the subarcuate fossa of the temporal bone, underneath the superior semicircular canal.

With an electrode placed on the round window of a cat's ear and sounds sent into the ear in the normal manner, the reproduction of the sounds from the round window were found to be a duplication of the sounds sent to the cat. This was the same for all tones and for different types of noises. Reproduction of the voice was accurate, but had a slight metallic ring to it. This pointed to the fact that the electrode picking up the phenomenon, from the round window, was obtaining not nerve impulses nor action currents, but an electrical response, similar to the telephone carrying conversation. Further investigation as to the cause of this cochlear phenomenon pointed to the Organ of Corti as its probable source. We have several lines of evidence that the Organ of Corti is the source of this response.

1. The vibration of the round window does not cause any such electrical waves reproducing sound. Experiments made on vibrating membranes with an electrode in contact with the membrane gave negative results.

2. In one experiment on a cat we obtained the cochlear phenomenon but no nerve impulses through the auditory tracts in the brain stem. Microscopic sections of the cochlea later showed the Organ of Corti to be normal, and that the spiral ganglion of the cochlea was the seat of an infection. This tended to prove that nerve impulses were not responsible for this source of sound reproduction.

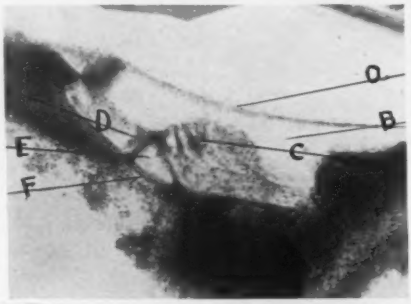

Fig. 1. Albino Cat. High power of cochlea, normal side. Showing Organ of Corti, Reissner's and basilar membrane. (a) Reissner's membrane; (b) Scala media; (c) Outer hair cells; (d) Inner hair cells; (e) Tunnel of Corti; (f) Basilar membrane.

3. Charles Darwin, in the Origin of Species, Prof. Alexander, of Vienna, and Dr. Stacey Guild, of Johns Hopkins, have reported that true Albino cats which have a blue eye on one side are deaf in the ear on the same side as the blue eye. We were fortunate to have an animal of this type in the laboratory. Our experiments with this animal showed that the cochlea on the side of the blue eye gave no response to any sound or tone stimulus. We also could find no nerve impulses in the auditory tracts coming from this ear. In the opposite ear we received normal responses from the cochlea and were also able to trace the auditory nerve impulses up to the medial geniculate body. On sectioning these two ears, the cochlea, Organ of Corti, spiral ganglion and the nerve fibres on the functioning side were found to be normal. On

the deaf side the Organ of Corti was not present, it had completely degenerated. Reissner's membrane had come down and fused with the basilar membrane of the membranous cochlea. The spiral ganglion of the auditory nerve had degenerated, and the auditory nerve also showed a marked degeneration of its nerve fibres. Therefore, the Organ of Corti must be present to get the cochlear response and the nerve impulses up the auditory nerve (see Figs. 1 and 2).

4. The question now arose whether changes of pressure or movement of fluid in the cochlea might be the cause of the response, since the scala media of the cochlea in the Albino cat was almost obliterated. Experiments done on normal

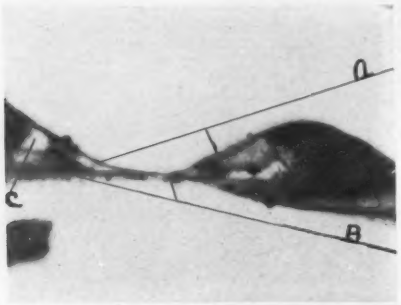

Fig. 2. Albino Cat. High power of cochlea, deaf side. Showing fusion of Reissner's and basilar membrane. (a) Reissner's membrane; (b) Basilar membrane; (c) Scala media.

guinea pigs showed a response from the cochlea similar to normal cats. A waltzing guinea pig (an animal similar to the waltzing mouse) was experimented on. These animals have degeneration of the semicircular canals, utricle, saccule and possibly the cochlea. Experiments with the animal gave no response from the cochlea. Microscopic sections of the cochlea of the waltzing guinea pig showed that the Organ of Corti was present; the scala media was normal; that is, Reissner's membrane was essentially normal in its relationship to the basilar membrane. The fluid was present in all three scalae. On closer examination of the Organ of Corti, evidence of degeneration of the sensory hair cells, particularly of the lower turns was found. The hair cells themselves were flattened out and in the upper whorls they showed some post-

mortem degeneration (see Figs. 3, 4 and 5). This final experiment showed that movement of fluid alone is not the cause of the cochlear phenomenon, and we conclude that the electrical waves are actually a true physiological response of the Organ of Corti.

We believe that the bending of the hair cells in the Organ of Corti sets up the electrical disturbance which reproduces so accurately the sound sent in. This electrical disturbance or "broadcasting" spreads throughout the whole cochlea and

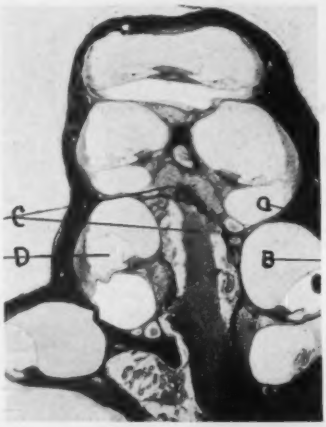

Fig. 3. Waltzing Guinea Pig. Low power of cochlea of waltzing guinea pig. Showing relation of three scalae to each other are essentially normal. Organ of Corti has a normal appearance in upper whorls. Organ of Corti degenerated in basal whorls. (a) Scala tympani; (b) Scala vestibuli; (c) Cochlear nerve; (d) Scala media.

is the phenomenon picked up from the round window. As mentioned before, if amplification is great enough, it can be obtained throughout the whole skull of the animal. These electrical changes which are set up in the hair cells of the Organ of Corti probably stimulate the nerve endings in these cells and initiate the nerve impulses which travel to the spiral ganglion and then through the auditory nerve to the auditory tracts in the brain.

On further experimentation with the cochlear phenomenon we examined the basilar membrane for a natural vibratory period. Examination of photographs of the waves repro-

duced by the cochlear response (picked up from the round window) showed that the basilar membrane had a natural vibratory period of its own somewhere between 800 and 1000. When a high tone is sent into the cochlea, about 2500, the basilar membrane vibrates for a very short interval of time,

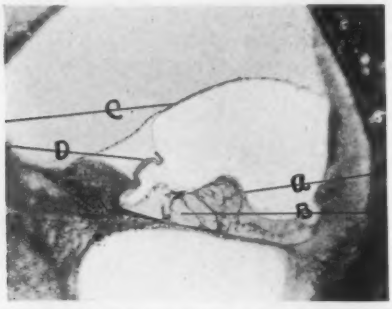

Fig. 4. Waltzing Guinea Pig. High power of cochlea, upper whorls. Organ of Corti shows degeneration but has normal appearance and relationship. (a) Organ of Corti; (b) Tunnel of Corti; (c) Reissner's membrane; (4) Tectorial membrane.

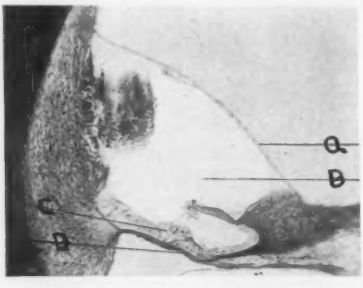

Fig. 5. Waltzing Guinea Pig. High power of cochlear basal whorls. Showing scala media essentially normal, degeneration of Organ of Corti, absence of Tunnel of Corti. (a) Reissner's membrane; (b) Scala media; (c) Organ of Corti; (d) Basilar membrane.

1/100 of a second, as its natural vibratory rate, meanwhile also gradually picking up and taking on the rate of the vibration sent in. That is, it vibrates between 800 and 1000 for a very small portion of time and then by degrees takes on the rate of the sound sent in, if at 2500, at 2500 per second. It was also noted that the higher the tone the smaller the wave response obtained. When a tone is sent in below 800,

it is found that the basilar membrane apparently vibrates as a whole, giving a very large response if the tone is loud. The evidence for this conclusion is somewhat complicated and we wish to repeat some of the observations before regarding it as a final conclusion. If we are correct, however, it means that the cochlea probably responds to sound in two ways. For tones below 800 to 1000, it may respond by vibrating as a whole. Above 1000 probably only a segment of the basilar membrane vibrates. To any sudden noise the first response is always the same: a brief quiver of the entire membrane.

We have evidence from the neuroanatomist that the cochlea has a double nerve supply. This has been reported by Dr. Lorente de Nó of St. Louis, and other investigators. The physicists who have been interested in hearing have also given evidence that the basilar membrane vibrates as a whole to low sounds or tones. Dr. Wegel of the Bell Laboratories, New York, has calculated that the basilar membrane vibrates as a whole up to 500 per second, but he concluded that it might be as low as 35 per second. We believe his first estimate to be more nearly correct. We have found in our work that it is probably higher in the cat, the vibratory point ranging from 800 to 1000.

There is also evidence of the double function of the cochlea from the comparative anatomy point of view. In an article read by the speaker at the American Otological Society in May, 1933, it was pointed out that the mammalian cochlea is the end development of a double organ for hearing, and that the probabilities are that the Organ of Corti functions in two ways, that is, responding as a whole for low tones and having specific resonant points for the high tones. The evidence at the present time from our experiments tends to the same conclusion. There is still much more work to be done on this, but as the evidence slowly accumulates we believe that it will develop a new theory of hearing.

When Wever and Bray reported their work, they assumed that what they heard through their apparatus was a pure physiological phenomenon and that they were picking up from the auditory nerve actual nerve impulses or, as the physiologist calls them when using an electrical apparatus for their detection, "action currents." As a result of this assumption they later published a theory of hearing, which they called

the Volley theory. This theory assumed, on the basis of known facts concerning other nerves, that a single auditory nerve fibre could only carry nerve impulses up to a certain frequency, for example—about 1000 nerve impulses per second. In order to explain the hearing of sound above 1000 vibrations per second, they assumed that more than one nerve fibre was concerned with the carrying of the impulses and that these impulses followed each other in a sequence so that five nerve fibres could carry a sensation of 5000 vibrations per second, each nerve carrying 1000 impulses at a time. If 20,000 vibrations, they assumed 20 nerve fibres carrying 1000 impulses at a time. These nerve fibres would not necessarily be stimulated all at the same time, but would pick up the stimulus at different intervals. Variation between the impulses of each nerve fibre would be that of the refractory period of the nerve, the refractory period being that portion of time in which the nerve cannot respond to a stimulus. This theory explains the Wever and Bray Phenomenon, but we do not think it is the true physiological explanation of sound perception.

The theory that we believe will evolve from this work is one that will probably combine the Resonance and the Telephone theories. The Telephone theory seems at present the most satisfactory for "low" tones, that is tones below about 800. While the Resonance theory seems well established for tones above 1500 vibrations. Between the two ranges lies a No Man's Land which may ultimately be claimed by either one or by both in combination.

The work at Johns Hopkins University, as previously stated, centered in the function of the middle ear. Dr. Crowe and his associates have claimed very definitely that increasing or decreasing the tension of the drum causes definite increase or decrease of hearing, that removal of the ossicles and also changes of tension of the tensor tympani muscle modify it. They also reported that the fixation of the round window by means of absorbent cotton or grafts on the round window caused an increase in hearing.

Culler, Finch and Girden, University of Illinois, in *Science*, Sept. 22, 1933, reported the experiments done on the function of the round window of dogs. They observed the electrical response and also tested acuity of hearing by the method of

conditioned reflexes. These experiments were done in trying to confirm the work at Johns Hopkins University. Their conclusions were as follows:

1. "That our gum-plugs, which meet the round window in direct apposition with no pressure, impede its normal oscillation and thereby impair acuity of hearing, as the accepted theory of cochlear function would lead one to expect.

2. "That actual hearing is affected in the same sense by our procedure as are the electric pulses which can be picked up from the cochlea and auditory nerve."

In other words, Culler and his associates believe that the experiments at Johns Hopkins University have not, at the present time, shown an actual increase of auditory nerve impulses going up the brain stem. The animal's perception of sound has not been increased in their experiments on the round window.

The death of the cat does certain interesting things to the cochlear phenomenon and to the auditory nerve impulses. When the cat dies the nerve impulses in the cochlear nucleus cease at once. The response received from the cochlea by means of the round window drops to 20 per cent or even as low as 2 per cent of its original intensity; that is, a loss of 80 per cent or more occurs within the first minute or so after the cat's death. After the cat has died the cochlear response can be obtained, diminishing in intensity for about thirty minutes to two hours. This residue is in all probability the result of the continuing life of the hair cells in the Organ of Corti, which can still set up an electrical response until they die.

Of what value has all this work been to the Otologist? There is sufficient evidence now to warrant that probably in the near future we will be operating on certain types of middle ear deafness to improve hearing; operations will be done on the middle ear mechanism chiefly, such as permanent perforations of the drum in order to allow sound to reach the round window or oval window more easily. The tensor tympani muscle will be cut to relieve marked retraction of the drum and thus allow the drum to transmit sound in a more normal manner to the cochlea. We believe the application of grafts for fixation of the round window will not be used.

The greatest value to the Otologist will be the placing of the theory of hearing on a firm foundation, thus enabling us to understand and to treat more intelligently, deafness occurring from various pathological conditions.

REFERENCES.

CROWE, S. J., and HUGHSON: *Arch. of Otolaryngol.*, Vol. 14, No. 5. Nov. 1931.

DAVIS, H., and DAVIS, P. A.: *Amer. Jour. Physiol.*, 101, 339, 1932.

DAVIS, H., DERBYSHIRE, A. J., and SAUL, L. J.: *Amer. Jour. Physiol.*, 105, 27, 1933.

DAVIS, H., SAUL, L. J., and LURIE, M. H.: *Trans. Amer. Otol. Soc.*, 1933.

DAVIS, H., and SAUL, L. J.: *Amer. Jour. Psychol.*, 45, 358, 1933.

HUGHSON, W., and CROWE, S. J.: *Ann. Otol., Rhinol. and Laryngol.*, 41, 332, 1932.

LORENTE DE NO: THE LARYNGOSCOPE, Vol. 43, pp. 1-38, Jan., 1933.

LURIE, M. H.: *Ann. Otol., Rhinol. and Laryngol.*, Dec., 1933.

WEGEL, R. L., and LANE, C. E.: *Phys. Rev.*, 23, 266, 1924.

WEGEL, R. L.: *Ann. Otol., Rhinol. and Laryngol.*, 1932.

WEVER, E. G., and BRAY, C. W.: (a) *Science*, 71, 215, 1930; (b) *Proc. Nation. Acad. Sci.*, 106, 344, 1930; (c) *Jour. Exper. Psychol.*, 13, 5, 1930.

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THE REGRESSION THEORY OF OTOSCLEROSIS.*

DR. LOUIS K. GUGGENHEIM, St. Louis, Mo.

May I first express my sincere thanks for this opportunity of telling you something of the work that is being done with otosclerosis at the Oscar Johnson Institute.

Otosclerosis was first described as a disease entity by Politzer. Since that time many investigators have carefully studied the histopathologic picture, have noted the later phases of the condition and have formulated certain theories as to etiology. Very noteworthy contributions have been made to science. However, we feel that not one of the investigators has taken into consideration all of the outstanding characteristics of otosclerosis. Furthermore, many of them have failed to evaluate properly such characteristics as they have noted. In the theory of regression we believe that all of these characteristics have been considered, and at least an attempt has been made to evaluate them. I would like to tell you of the work of Wittmaack, Bruhl, O. Mayer, Weber and the rest, but you are thoroughly familiar with these theories, and furthermore the lack of time makes it necessary for me to confine myself to the theory of regression.

What are the outstanding characteristics of otosclerosis? First, the condition is hereditary, with a genetic formula of two dominant factors, one sex-linked; secondly, otosclerosis is usually bilateral, and very frequently bilaterally symmetrical. Third, there is a site of predilection, anterior to the oval window, in the region of the fissula ante fenestram; next, the three structures which are usually involved, the oval window, the round window and the cochlea, are recently acquired structures; next, otosclerosis may involve the footplate of the stapes, but it does not involve the crura.

Let us now attempt to evaluate these various characteristics. In dealing with an hereditary affection, we would like to find somewhere in the developmental period an anlage or tissue

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abnormality which would indicate the future osseous dystrophy. This should be possible, with an adequate technique, and a complete knowledge of the morphology of the embryo throughout the developmental period and extending to the second year of life when the ear is practically complete. There are changes later, of course, but it is practically complete by that time. This is a very big order, because I do not know of anyone who knows the morphology of the ear from the beginning straight through to the second year, that is, who knows the morphology plus the cytology. The general morphology is generally well understood, but the cytology is not yet thoroughly worked out. There are many, many years of work to be done. We must then have an understanding of the ear, particularly from the 28th day, when the auditory vesicle is closed off from the ectoderm and is surrounded by condensed mesenchyme. The condensed mesenchyme is the first step in the formation of the capsule. When we come to the second month, we must be thoroughly familiar with the mesenchymal tissue surrounding the precartilaginous capsule, which is highly vascular, and contains chondrogenic cells which are differentiated mesenchymal cells with large round nuclei and many protoplasmic processes. Some of these processes disappear; others extend around and coalesce with one from the other side, forming in this way the cartilage lacunae. The matrix, which is fibrillar in the beginning, later becomes homogeneous.

(All points were illustrated with slides.)

Under higher magnification, we see the semicircular canal surrounded by a syncytial reticulum. When this is resorbed, we have formed the perilymphatic spaces of the labyrinth.

When we reach the third month the precartilage has changed into pure cartilage and a true perichondrium has appeared. At the fourth month, the very interesting process of ossification is well under way. Owing to the presence of phosphates at this period, there is a deposit of calcium in the cartilage. Then the cartilage lacunae become swollen and we see an invasion through perichondrial ruptures of osteogenic buds accompanied by osteogenic cells and histocytes. At four and one-half months, periosteal bone has been laid down. At this period we note certain cartilage rests. These are, I believe, calcified lacunae. The cartilage cells are destroyed by the osteoblasts which have invaded them. Then we have the osteo-

blasts in epithelial alignment which lay down the enchondral bone.

When we reach the seventh month, you note that the periosteal bone is now composed of interlacing trabeculae; enchondral bone is far along in its development, with cartilage rests surrounded by thick trabeculae. (Here was demonstrated slide showing calcified cartilage rests, marrow spaces, osteoblasts lined up along the edge, web bone being laid down, osteoblasts becoming osteocytes. Within are cartilage rests surrounded by osteoid tissue.)

At two months of age the cartilage rests are still noted. There are wide trabeculae of bone and the marrow spaces are decreased in size.

In the adult we note the enchondral bone, the heavy periosteal bone and the endosteal bone surrounding the membranous labyrinth. In the enchondral portion we find the fine fibered lamellarless bone, lamellar bone around the Haversian canals and the rich content of cartilage rests. In otosclerosis the latter disappear. In the adult we find also undifferentiated mesenchymal cells in the marrow spaces. The undifferentiated mesenchymal cells make it possible for otosclerosis to be initiated.

Our studies through the developmental period were rewarded last June by the finding, in a seven months' fetus, of an osseous dystrophy which we believe is otosclerosis. The rest of this embryo is beautifully normal. In the round window region you see blue staining areas. The rest of the bone is all pink or red. You see in the wall of the niche these dark areas which are web bone. The remainder is of the normal structure of enchondral bone. (Demonstration of slide showing a different staining to make the web bone stand out.)

(Demonstrating a normal area of enchondral trabeculae and cartilage rest), I would like you to look upon this as a garden. The fine fibered structure we can compare to the soil. The osteocytes in their beautiful normal arrangement are the flowers. The osteoblasts are lined up, laying down the enchondral bone. This is a normal portion of the labyrinth. Another slide with different staining shows the large amount of cytoplasm and large oval nuclei of the normal osteocytes. We see periosteal bone, marrow and trabeculae of enchondral bone. We come now to these dark areas which we believe to

be otosclerosis. The first thing we notice is that in the marrow spaces, instead of a rich content of bone forming cells and hematopoietic elements, there is a mass of fibroblasts which are developing from the mesenchymal cells, with the formation of connective tissue which immediately becomes calcified. All of these dark areas are blue staining web bone which is not a normal thing. In addition you will note that throughout the enchondral bone there are blue masses. The osteocytes are changing, the nuclei becoming pyknotic and the cells being of abnormal shape and in clumps. I mentioned that the later phases of otosclerosis had been observed, because this, the earliest phase, has never before been seen. (Demonstration of slide with different staining of the same area in order to show the changes in the osteocytes, etc.) Halisteresis is going on; calcium is being removed from the bone. Blue staining web bone is forming in the degenerating enchondral trabeculae.

We come now to a most interesting part of this specimen. The masses of cartilage rests in these dark staining groups are completely inundated by the blue staining web bone. Normally they are surrounded with osteoid. Note the degeneration of the ossified lacunae (cartilage rests). The degenerating bone is being resorbed. Note that the osteoblasts which were lined up to form bone have been interrupted in their work and are falling off into the marrow spaces. They are no longer able to stick in their places. This weed-like otosclerotic bone destroys everything in its path. You can note here the distinct outline of the cartilage elements which are being overwhelmed and destroyed by this process. (Demonstration of slide showing how the cartilage rests disappear.) We know that in otosclerotic foci (second and third stages) we do not find cartilage rests, but we never before knew when or how they disappeared. Again we see an area of this blue staining web bone and here a portion of the normal enchondral labyrinth which is alive (part of the garden undisturbed by weeds). Note the difference between the normal bone cells and the osteocytes in the diseased tissue. The blue web bone grows right into the enchondral bone.

We have examined very carefully the site of predilection. We see it here, just anterior to the footplate of the stapes; a very intriguing area. There are cartilage cells with osteoid around them, and a certain amount of blue staining fibers.

In this area, instead of the normal marrow we see mesenchymal cells and fibroblasts. We would like to call this an early focus of otosclerosis. Dr. Marvin Jones has a specimen like this which I saw yesterday. It is normal. We find it normally in the fetus. The same findings around the fissura ante fenestrum are also normal and not otosclerosis. I have examined sixteen fetuses of the same age as this rare one and in none has anything suggesting blue web bone been found. So we feel that the blue dystrophy in our case is a very early otosclerosis.

We come now to the second point, that otosclerosis is usually bilateral and very frequently bilaterally symmetrical. To the biologist that means that it is a gene propelled thing and not a disease in the ordinary sense. It is very hard to fit those findings into any disease process occurring after birth.

The third point is that anterior to the oval window is the site of predilection in otosclerosis. This is the region of the fissura ante fenestrum, which is an interesting passageway leading from cisterna to tympanum. We see it here in the four and one-half months' fetus passing through the cartilaginous capsule into the tympanum. Here we have it at seven months. It is filled with young connective tissue and lined with cartilage. At 13 months we see the footplate of the stapes and the fissura ante fenestrum passing from the vestibule outward. The content of it is mesenchymal cells, histocytes, osteogenic cells, fibroblasts and connective tissue. According to T. H. Bast, in a recent issue of the *Archives*, there are three theories as to the origin of the fissura. Mayer and Kosokabe believe it a synchondrosis; Perozz', that it is a growth area for the cochlea. The third theory which originated at our Institute is that the fissura ante fenestrum is the homologue of the accessory endolymph duct of the acanthius vulgaris, raja clavata, and the homologue of the ductus fenestrae ovalis of the rana esculenta. The possibility that the fissura ante fenestrum is a vestigial structure, and that otosclerosis starts in its immediate vinicity as a site of predilection, suggests that the genes for this osseous dystrophy we call otosclerosis, are genes of regression. The fissura has not been found often in the intermediate phyla between fish and man. (Hyrtl found it in the hyena and antelope.) However, I have from Professor Hansen, biologist of Washington

University, that even if it had not been found in the intermediate phyla, it can still be a homologue of the lower structures, as biologists believe that man is not derived from the intermediate phyla of today, but from a very much lower reptilian amphibian form no longer in existence.

(Demonstration of picture showing the ductus fenestrae ovalis of the frog, passing from the perilymphatic space through the capsule to the tympanum and ending as a blind sac.)

The oval window makes its appearance first in the clupea harengus (herring) from the incomplete articulation of epoticum, opistoticum and occipitale basilaris. In acanthous vulgaris there is a membrane covered oval window; also in the raja clavata. Not until we reach the stage of the amphibian do we see an oval window which is covered with a bony operculum. This is in the proteus anguinus. There is no annular ligament and no movement. In the menopoma alleghaniensis there is an oval window with a columella, footplate and annular ligament, and there is movement. Not until we reach the frog do we have a round window. As for a cochlea, we have nothing comparable to the mammalian cochlea until we reach the reptiles. The elongation of the pars basilaris of the amphibian produces the cochlea of higher organisms.

The next point: The three structures commonly involved in otosclerosis, oval window, round window and cochlea, are recently acquired structures. We believe otosclerosis is a variation in these structures; not accidental, but due to the presence of genes of regression. At the site of predilection, anterior to the footplate, we see this focus of otosclerosis. This is the second stage. If this focus replaces the footplate the oval window is closed. When the round window is involved we may have a closure. When there is involvement of the internal auditory meatus, the cochlea may be surrounded and its function destroyed. (Demonstration of specimens showing foci at round window and cochlea.) When these three things occur, the oval window closed, the round window closed and the cochlea's function destroyed, we see produced a morphology which is comparable to that of the lowest vertebrates. In the myxine glutinosa, the lowest vertebrate, we see the ear as a closed cartilaginous affair. There are no windows and no cochlea. The labyrinth consists of utriculus and two

canals. In the next higher form, petromyzon fluviatilis, we again have a cartilaginous capsule closed; a sacculus has evolved. Next in the acipenser sturio we have the addition of a third semicircular canal and the formation of an outpouching of the sacculus, the lagena cochleae; the capsule is closed. So, in the process of otosclerosis we have the production of a morphology which suggests that of the lowest vertebrates. The genes that produce otosclerosis, we believe, originated in the fish period, and in the human are carrying out the normal expression for their particular period. They are trying to produce a capsule for their period which in the human results in disease.

We now come to the last point, that the crura of the stapes are not involved, only the footplate. I have never seen that mentioned except in the article by Lenge in Henke and Lubarsch. He does not analyze it or even discuss it. It is simply mentioned as a fact. It suggests immediately to us that there is some difference between the crura and the footplate. Why should not otosclerosis involve the crura of the stapes? Some years ago observations were made at our Institute that the footplate of the stapes is derived from the capsule of the labyrinth instead of from Reichert's cartilage, as is stated in all modern textbooks. The crura are derived from the cartilage of the second hyoid arch. This was published as an original observation. After publication it was found in a very old book that the same observation was made in 1879 by Gradenigo. The footplate being from capsule, it returns, in otosclerosis, to the tissue whence it was derived.

At two and one-half months we see the stapedial ring composed of pure cartilage and the oval window covered by a mesenchymal curtain in apposition to the ring, but not at all connected with it. At three months we see the ring is flattened out medially in order to accommodate itself better to the oval window curtain. At four months the ring is fused with the footplate, although you can still see the difference in structure (footplate is of younger cartilage). Under higher magnification you can see plainly the difference in texture. At four and one-half months it is difficult to discern, but you can still see the difference in texture between the footplate and the crura. The crural cartilage is much older. At two years you can no longer discern any difference. In the adult

of course there is no difference at all in appearance. When the footplate is displaced by otosclerotic bone the capsule is closed; the morphology of the lowest vertebrates is reproduced.

There are numerous instances in the literature of vestigial structures in human ears, such as Fischer's case of a third macula and reptilian cristae; Ruttin's third macula, etc.; but as far as regression is concerned, we know of no other instance than otosclerosis. In the lower animals there are some very interesting examples of regression. In the very young whale embryo there are several tubercles around the external auditory meatus. In the older embryo these have fused into one. At birth there is no sign of an external ear. This is regression. Perhaps the most interesting of all is the case of the ascidian or sea squirt. In the larval stage this little organism has a tail and swims freely; a brain, one eye and one ear, and tactile organs. (Demonstration of slides.) The ear consists of a single cell almost completely filled with an otolith, and we see only an edge of cytoplasm at the periphery of the cell. In a few days the organism loses its tail, the eye and ear disappear, the brain disappears, and it attaches itself to a rock by means of its tactile organs. It may live like that for a year or longer, is bisexual, sessile, has no brain, eye or ear.

I have shown you then in the seven months' fetus what I consider to be the earliest stages of otosclerosis. The second stage you are all familiar with. (Demonstration of slides.) In the cases we ordinarily see there are areas of resorption in the capsule, which are then filled in with blue staining web bone. In the earliest phase you have seen phenomena which are different from anything ever before seen in otosclerosis. In the third and last stage of otosclerosis, after many years of pathological new bone formation, we get a very dense structure; the marrow spaces are few and small. Blood vessels disappear and the dense bone stains red.

In conclusion we believe that it is possible that otosclerosis is due to the presence in the human ear of fish genes which produce a secondary activity after the genes for normal structure have expressed themselves, and that the lower vertebrate genes express themselves secondarily by the production of this web bone in an attempt to form a capsule of their own period.

Missouri Theatre Bldg.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of January 17, 1934.

(Continued from a previous issue.)

DR. WESLEY C. BOWERS: In discussing this interesting subject, so ably presented here tonight, I feel it would be well to emphasize several points of great importance. There are of course two classes of failure—those from the patient's standpoint and those from the surgical standpoint.

Failures from the patient's standpoint are just as important—perhaps more so—than the surgical failures, as they are far more common and consequently have had a great deal to do with causing the dissatisfaction we hear expressed by the general profession and the layman. All too often the patient does not fully understand the object of the operation and the results which are to be expected. He may think that the removal of his tonsils will cure his rheumatism. When, perhaps after a considerable time the rheumatism recurs, he feels that the tonsillectomy was a failure. Another patient may develop lymphatic tissue in the tonsillar fossae. A general practitioner, unable to differentiate between lymphatic tissue and tonsillar tissue, may tell such a patient that his tonsils have grown back. The patient and the practitioner consider the operation a failure. Again, no matter how perfect or how lasting a result we may obtain from tonsillectomy, surgically speaking, there will be times when a little lymphatic tissue will appear in an otherwise smooth fossa; this tissue may develop infection and have to be removed. The surgeon who did the perfect operation is liable to censure by patient and practitioner unless the discoverer of the lymphatic tissue is fair enough to defend him, or the operator has taken a few moments beforehand to tell the patient of this and other possibilities following his operation.

From the surgical standpoint, attention to details is of major significance in tonsillectomies. Failure to remove the plica triangularis and the lymphatic tissue at the base of the tonsillar fossae may result in failure to relieve the patient of the entire focus of infection, even though the tonsillectomy be complete. On the other hand, in our anxiety to remove all lymphatic tissue at this point, we must not leave a raw surface on the tongue opposite the base of the tonsillar fossae, as when this occurs we get a shortening of the anterior pillar with adhesions between the tongue and the base of the tonsillar fossae. Such a complication causes severe discomfort to the patient for an indefinite period. The way to avoid leaving a piece of tonsil in a fossa is to carefully examine the capsule, making certain that it is intact; otherwise you are not sure that the tonsil is completely removed, no matter how clean the fossa may look.

In regard to failures in sinus surgery—there is probably no other branch of surgery which has been as severely criticized and misunderstood by both laity and the general profession.

(1) To avoid improper education of the general profession and laity: If we feel that an antrum should have a window made into it, we should not leave the patient under the impression that this will cure the sinus immediately. In this operation we do not remove all the disease, we merely give Nature a better chance by increasing drainage and ventilation. In time Nature will

cure most of these cases. If the membrane is so badly diseased that nothing but its removal will cure it and we decide on a radical procedure, we should not lead the patient to believe that this operation will cure his entire nasal sinus condition unless—as rarely happens—there is no other sinus involved. If we do a Lynch operation on the frontal, do not let us lead the patient or the general profession to infer that such a procedure will for all time prevent the possibility of reinfection of the frontal. If it is necessary to operate on a sphenoid, let us not be too sure that this particular sphenoid is a simple structure. It may have an exceedingly thick wall with the presence of an osteitis—which will mean a continuation of the pain. It may extend to or connect with the opposite side; the opposite sphenoid may extend to the side you are operating on; or existing ramifications may be such that it is impossible to get proper drainage. Again, let us not lead the general profession or layman to think we are cleaning out all the ethmoids when we do an internal ethmoidectomy.

(2) From the surgical standpoint, the most important cause of failure is improperly selected operations, the result of bad judgment due to lack of knowledge and experience. If we operate on a septum and remove the anterior ends of both middle turbinates in an attempt to cure stuffiness and discharge, when we also have present two chronically infected antra, we are, as you know, doomed to failure, even though the surgical results be perfect. Yet this choice of operation occurs far too often. If we operate on a nose without due consideration of a coexisting allergic condition, endocrine imbalance, blood dyscrasia or other constitutional weaknesses, we are defeated before we start. Most failures in sinus surgery are due to lack of thorough study, failure to consider all coexisting conditions, lack of mature judgment as to the correct operative procedure and the difficulties encountered, some of which are impossible to surmount.

In speaking of failures in simple mastoidectomies, I would like to stress the importance of selecting the proper time for operation. There is a time in the early period of all involvements of the mastoid before nature has had time to wall off the infection when it is best to defer operating in so far as other conditions warrant. Again, there comes the time when waiting involves grave chances. To be able to determine just when to wait and when to operate requires the nicest of judgment, and is the deciding factor in many cases.

Study and examination of mastoid and petrous bones leads one to wonder that infection at these points ever gets well. We do the most we have any right to do, follow all cells to the limit of safety; even then we may fail because some cells further in refuse to drain through the openings we have made. In which case, faced with threatening symptoms, we go in again to be certain we have overlooked nothing around the semicirculars or further above the middle ear. Finding nothing, we do a radical and search carefully for cells in any part, particularly around the superior semicircular, the Eustachian tube or below the promontory; this failing, we are faced with the problem of boldly approaching the top of the petrous bone by means other than following leads. It is sometimes very difficult to find such leads. I have occasionally packed off these parts with adrenalin for a time, while finishing up some part of the wound; this will sometimes result in sufficient shrinkage to allow a tiny bead of pus to force its way through the opening which is in itself so small that it will admit with difficulty the smallest middle ear probe; this opening can then be enlarged sufficiently to obtain free drainage, which may be maintained by means of a rubber tube.

I would like to mention three points which I believe are common causes of failures in radical mastoidectomy: (1) Too small a meatus is a great disadvantage and a frequent cause of failure. (2) Improper after care is another common cause of failure. (3) The literature is full of discussion about the failure to close off the Eustachian tube, which is supposed to result in discharge from this region. I feel that it is not a question of closure of the

tube, but the presence of infected cells around it or possibly the presence of a petrousitis which is draining into this area.

DR. WILLIAM H. TURNLEY: Mr. Chairman, ladies and gentlemen: I am here to report on the effect of tonsillectomy in rheumatic cases in conjunction with the paper by Dr. David Jones. The term rheumatism includes all painful conditions of the joints, muscles, fascia, bones and nerves as described by the patient as "rheumatism." These are all ambulatory cases.

During the year 1931 there were 721 patients out of the 11,925 operated upon who gave a history of rheumatism, representing 6 per cent. In 1932, there were 76 out of 12,001, or again 6 per cent. In 1933, there were 694, or 6.5 per cent out of 10,583 operations.

As has been reported, during the three years there were 34,509 operations, of which 28,960 returned for a postoperative inspection. But after a few years have passed they are a little more reluctant about returning for a check-up on their rheumatism. Only about 10 per cent of those to whom we sent cards returned; consequently, this series represents only 300 cases so far. And of these, 66 per cent were women and 34 per cent men. There were 33 per cent Hebrews, only 4 per cent colored, and the remaining 63 per cent included a very noticeable proportion of Porto Ricans. There were no Chinese. About 6 per cent of the total had had acute rheumatic fever.

These patients gave a history of rheumatic pains at various times, intermittently from 3 weeks to 25 years. Many reported an associated tonsillitis, while a great many reported no tonsil trouble at all. It has been observed also that the rheumatic type of tonsil is not the large follicular tonsillitis with pus, but rather the small obscure tonsil which has given very little trouble except for a thin secretion which trickles down the back of the pharynx.

Their ages varied, the youngest being three and the oldest 59 years of age. Of those from three to ten years, there were 7 per cent; from ten to 20 years old, 10 per cent; from 20 to 30 years, there were 15 per cent; from 30 to 40, there were 37 per cent; from 40 to 50 years, 23 per cent, and from 50 to 59, there were 8 per cent. By far the greatest per cent are from 30 to 40 years of age.

As to results, it was difficult to figure for each age the percentage "cured," "better," "unimproved" or "worse," but the average was about 83 per cent giving a history of either no further trouble or definitely better. About 12 per cent said that they had noticed no difference and about 15 per cent said they were worse. The younger the case and consequently the shorter the duration, the more marked was the improvement, and vice versa. But of those cases that gave a history of no improvement, most all of them either had some complication or other foci of infection, chiefly a sinusitis. Another important observation was the fact that 90 per cent said they had fewer colds and sore throats since the operation.

Rheumatism, I think, is one of the great medical problems of civilization, especially in the temperate zone, and that rheumatism is in some mysterious way related to focal infection, but that focal infection does not explain the entire problem, is my firm belief; also, that the tonsil, due to its direct communication to the lymphatic system, through its characteristic crypts, is probably the greatest source of infection. I therefore advocate the removal of tonsils in all rheumatic cases until the etiology of rheumatism is better known.

Mr. Chairman, I wish to express my appreciation to Dr. David Jones for having me placed on this program and to the rest of the ladies and gentlemen for listening.

DR. SAMUEL J. KOPETZKY: It is manifestly impossible in the time at our disposal to call upon all the men here who have had experiences by which others might profit. If there is anyone who has an unsolved problem, if he

will ask the question and it naturally falls in the category of any of the essayists, if any of the essayists is able to answer the question, he will do so.

DR. JOHN M. LORE: Does Dr. Jones think that an injury to the 9th nerve, in going after the tonsil, has anything to do with the change of taste or complaint of metallic taste after operation?

DR. ARTHUR J. HERZIG: I should like to ask Dr. Jones whether, in these cases of so-called recurrence of the tonsils, the tissue is examined microscopically, to see if it is tonsillar tissue or granulation tissue? It is probably the latter, as I have never seen a tonsil grow again if properly removed.

DR. SAMUEL J. KOPETZKY: I will answer my good friend Dr. Kernan. I think in competent hands the simple mastoid operation is fool-proof. In reference to taking pictures of the petrous bone in acute otitis, I believe in taking a plate showing the petrous bone. It incidentally shows the mastoid process too. I believe in doing this in all cases of acute otitis in which there is a possibility that you may have to do some surgery so as to establish two facts. First, it shows what type of temporal bone you are dealing with; and second, is or is not there a shadowing of the temporal bone and petrosal pyramid, because the involvement of the petrosal pyramid is a complication not *of mastoiditis*, but *of otitis*? Dr. Bowers in 1928 reported on two cases before this Section, I believe. The little leakage from the petrosal of which Dr. Bowers speaks is one of the proofs that this is a complication of otitis, not of the mastoiditis. It sometimes plays its rôle in those cases which do not resolve.

THE NEW YORK ACADEMY OF MEDICINE.

Combined Meeting of the

SECTION OF OTOLARYNGOLOGY

AND

SECTION OF PEDIATRICS.

February 21, 1934.

Report of Two Cases of Pharyngeal Hematoma with Fatal Hemorrhage from Retropharyngeal Abscess. Dr. William Spielberg.

Case 1. A boy, white, age 8 years, was admitted to the Beth Israel Hospital on the service of Dr. Kopetzky on Feb. 26, 1933.

Chief complaints on admission: 1. Right sided headache. 2. Right side of neck swollen and painful for four weeks. 3. Difficulty in breathing.

Family and previous history: Negative. Tonsillectomy at the age of 3 years.

Present Illness: Began suddenly about five weeks before admission to hospital, with slight chill, dizziness and elevation of temperature to 100-101° F. On the third day a swelling appeared in the right side of the neck, accompanied by a constant severe right sided headache. The glands in the neck got smaller and at times larger. On the seventh day of his illness, the family physician noticed a large swelling in the right side of the throat. However, since the patient was comfortable he was left alone and by the end of about three weeks all his signs and symptoms subsided. Three days before admission to the hospital the swelling in the neck and throat reappeared. The patient felt ill again, was unable to swallow and complained of a severe pain in the neck and throat. On several occasions he coughed up blood or blood stained mucus, and complained of difficulty in breathing.

On careful examination the boy did not look ill, was a little restless, sat up in bed, took nourishment fairly well and talked with somewhat of a muffled voice.

Physical examination revealed a congested right ear drum and slight tenderness over the tip of the right mastoid. The nose was normal. The throat was moderately congested. No tonsils were present. Occupying the right hypopharynx, outer wall and extending into the epipharynx and pushing the soft palate forward was a large, smooth and very tense mass. It was not painful on pressure. A small mass of enlarged cervical glands were found in the right side of the neck at the angle of the jaw. All other findings were normal.

Temperature, 99.2; pulse, 110; respiration, 26. Blood count: red blood cells, 3,800,000; hemoglobin, 75 per cent; leukocytes, 24,000; polys., 84; monos., 16; staffs, 2; segmented, 82; large monos., 16.

A diagnosis of right retropharyngeal abscess extending high up into the epipharynx with secondary cervical adenitis was made and the patient was taken to the operating room for evacuation of pus from the abscess.

Operation: Examination on the operating table without anesthesia revealed a large tumor-like fluctuating mass in the pharynx on the right side, pushing the

pillars of the tonsils forward and impinging upon the soft palate, completely filling the nasopharyngeal space.

Selecting an area behind the posterior pillar and directing the instrument toward the pharyngeal wall, an incision was made and a moderately large, old organized clot was evacuated. Widening of the incision was followed by an uncontrollable hemorrhage so profuse and overwhelming that within less than a minute the patient became exsanguinated and ceased breathing.

Following the tract in the pharynx, it seemed to lead to the base of the skull. Another perforation leading from the cavity in the mass toward the median line high up in the pharyngeal wall was found postmortem. An incision into the neck was made, the jugular vein inspected and found intact. Dissection along the internal carotid was continued to the pharynx until an opening was made connecting with the tumor mass and no evidence of a perforated blood vessel found.

Postoperative Diagnosis: Dissecting hematoma of the posterior pharyngeal wall possibly due to the erosion of a branch of the internal carotid artery.

Pathologic examination by Dr. Plaut of the specimen taken from the operative area in the pharynx and neck showed: Gross: Two lymph nodes. Microscopic: Lymph nodes with large germ centers. Tissue spaces in the capsule and in the connective tissue are engorged with mononuclear elements. No leukocytic infiltration found.

Comment: The tenderness of the tip of the mastoid associated with the intense right sided headache may lead one to speculate on the possible presence of a periphlebitis or phlebitis of the jugular vein and lateral sinus.

In this case undoubtedly the blood tumor was the result of a retropharyngeal abscess. The latter resolved but caused an erosion of a branch of the carotid artery and formation of a blood tumor, the blood tumor being mistaken for a retropharyngeal abscess.

We feel that by preliminary puncture and aspiration a correct diagnosis might have been made and a catastrophe avoided.

Case 2. A boy, age 6 years, was admitted on April 11, 1933.

Chief complaints on admission: Pain in right side of throat for four days. Family and previous history: Negative. Tonsils removed at the age of 18 months.

Present Illness: Had measles four weeks prior to admission, from which child fully recovered. Four days before admission to hospital the child suddenly took ill with a temperautree of 104° F., and severe pain in right ear. A swelling was noticed in the right side of the neck. Vomited several times; no blood. The child was able to swallow only liquid food with considerable pain.

Physical examination revealed a restless child, who appeared acutely ill and in pain. Examination of ears revealed a moderately congested right drum with no obliteration of land marks. There was no tenderness of the mastoid. The nose showed moderately congested inferior turbinates with mucopurulent secretion in both nares. The throat showed diffuse congestion of the pharynx and a small indurated mass in the right side of the hypopharynx in back of the posterior pillar. The child's tonsils were out. Moderately swollen cervical glands in the right side of the neck. Other physical findings were normal.

Temperature, 102.2° to 104.2°; pulse, 130; respiration, 30. Blood count (on admission): red blood cells, 4,000,000; hemoglobin, 72 per cent; leukocytes, 21,500; polys., 81; monos., 19; staff, 15; segmented, 64; eosin., 1; small mono., 8; large mono., 11; baso., 1.

A diagnosis of right retropharyngeal abscess was made, but due to the

marked induration of the mass and absence of fluctuation, it was decided to wait before incising.

Operation: On the third day after admission the child was taken to the operating room and the abscess was incised, the opening enlarged with forceps. No pus was found; only a small quantity of bloody fluid evacuated. The patient was to his bed and condition observed. During the next two days the retropharyngeal swelling subsided, but the glands in the neck became larger and showed fluctuation. The fluctuating area was incised externally and pus evacuated. The patient did very well until the fourth day following operation on the pharynx, when he suddenly began to bleed profusely from the mouth. The patient was rushed to the operating room, where, under a general anesthetic hemostasis was applied and the bleeding area sutured. The child, however, had lost a considerable amount of blood and saline was given intravenously and later followed by transfusion of 200 c.c. of blood. The pulse and general condition improved and the child was returned to the ward. During the next 24 hours the patient did not talk and at times showed Cheyne-Stokes respiration. On the fifth day postoperative, a left sided hemiplegia was noticed. The neurologist saw the patient and made a diagnosis of left flaccid hemiplegia, due probably to an embolic lesion involving the right hemisphere, probably subcortical, involving the middle cerebral artery. The source of the embolic lesion was most likely from the necrosing arterial site. Dr. E. D. Friedman, neurologist of the hospital, also diagnosed the condition as a hemiplegia due to an embolic focus in the right hemisphere. The patient lingered for two more days and died.

Comment: In this case the negative findings on incision indicate that the retropharyngeal abscess was incised prematurely. The infection and necrosis extended to and caused an erosion of a branch of the carotid artery with subsequent hemorrhage and embolus to the brain.

Hemorrhage from Pharyngeal and Peritonsillar Abscess. Dr. Samuel Salinger.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Results of Tonsillectomy in Private Practice. Dr. M. H. Bass.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Personal Observations on the After-Effects of Tonsillectomy. Dr. Howard H. Mason.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

A Practical Consideration of the Nasal Accessory Sinuses in Children. Dr. Wm. Mithoefer.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

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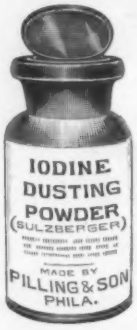
IODINE-BORIC ACID DUSTING POWDER-SULZBERGER



Very effective in chronic suppurative infections of the middle ear, and in nasal sinus infections. (See The Laryngoscope, June, 1930; reprint on request.)

An intimate association of iodine and boracic acid, prepared under process of Dr. Nathan Sulzberger, of New York, offering free iodine in **minute division, without the necessity of breaking up a chemical iodine compound.**

Made in two strengths of iodine; the strong approximately two per cent (2%), the weak approximately one per cent (1%). Both strengths should be available. Treatment should be started with the weak powder.

PRICES

☐ 1 oz. 1%..... $1.50
☐ 1 oz. 2%..... $1.50

[Unless you have an account, we will send these small items C. O. D. It saves a great deal of bookkeeping and postage. Or remit with order.]

The George P. & Son Co. PILLING Arch & 23rd Sts. Phila., Pa.